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Original Article

Social Determinant Pathways to Hypertensive Disorders of Pregnancy Among Nulliparous U.S. Women

Monica H. Keith, PhD [a,*,1], Melanie A. Martin, PhD [a,b]

[a] *Department of Anthropology, University of Washington, Seattle, Washington*
[b] *Center for Studies in Demography and Ecology, University of Washington, Seattle, Washington*

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A B S T R A C T

Background: Hypertensive disorders of pregnancy are a leading cause of maternal morbidity and mortality in the United States and impact Black mothers at disproportionately higher rates. Hypertensive disparities among racialized groups are rooted in systemic inequalities, and we hypothesize that clinical markers of allostatic load capture embodied disparities in stressors that can link upstream social determinants of health with downstream hypertensive outcomes.
Methods: We analyzed observational cohort data from the Nulliparous Pregnancy Outcomes Study: Monitoring Mothers-to-Be ($n = 6{,}501$) and developed a structural equation model linking latent social determinants of health, longitudinal markers of allostatic load across gestation, and hypertensive pregnancy outcomes in a multigroup framework.
Results: Non-Hispanic Black mothers-to-be ($n = 1{,}155$) showed higher rates of hypertensive disorders of pregnancy (32%) than non-Hispanic White women ($n = 5{,}346$, 23%). Among both groups, the social environment showed stronger direct effects on allostatic markers than via behaviorally mediated dietary, exercise, or smoking pathways. Demographic aspects of the social environment (e.g., household income, partnered status) were the most salient predictor of hypertensive risk and showed stronger effects among Black women.
Conclusions: Embodied stress rooted in the social environment is a major path driving maternal hypertensive disparities in the United States, with effects that vary across racialized groups. These pathway findings underscore the greater impact of systemic stressors relative to individual health behaviors. More comprehensive and detailed analyses of sociostructural domains are needed to identify promising avenues for policy and intervention to improve maternal health.

Hypertensive disorders complicate up to 20% of pregnancies and are the leading cause of maternal morbidity and mortality in the United States (Centers for Disease Control and Prevention, 2022; Collier & Molina, 2019; Ford et al., 2022). Hypertensive disorders of pregnancy (HDP) associate with a range of adverse outcomes, including preterm birth, placental abruption, renal failure, stroke, long-term cardiovascular morbidity, and maternal mortality (Butwick, Druzin, Shaw, & Guo, 2020; MacDorman, Thoma, Declcerq, & Howell, 2021; Ying, Catov, & Ouyang, 2018). Both preeclampsia and gestational hypertension have increased in prevalence over the last several decades in the United States, with higher burdens among Black mothers (Ananth, Keyes, & Wapner, 2013; Miller et al., 2020; Tanaka et al., 2007; Wallis, Saftlas, Hsia, & Atrash, 2008). Although social and structural determinants of health have gained recognition as drivers of disparate health risks (Crear-Perry et al., 2021; Howell, 2018; O'Reilly, 2020), few studies have modeled these as root causes of racialized/ethnic inequities along pathways with other risk factors (Bagby, Martin, Chung, & Rajapakse, 2019; Dunkel Schetter et al., 2013; Ramey et al., 2015; Saluja & Bryant, 2021). In this analysis, we aim to link pathways across multilevel social determinants of health (SDOHs), clinical markers of allostatic load, and hypertensive pregnancy outcomes among a national cohort of nulliparous women in the United States using structural equation modeling (SEM).

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* Correspondence to: Monica H. Keith, PhD, Department of Anthropology, Vanderbilt University, 124 Garland Hall, Nashville, TN, 37235. Fax: (615) 343 1444.
E-mail address: monica.h.keith@vanderbilt.edu (M.H. Keith).
[1] Present address: Department of Anthropology, Vanderbilt University, Nashville, Tennessee.

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Clinical risk factors for HDP include prepregnancy overweight or obesity, type 1 or 2 diabetes mellitus, chronic hypertension, nulliparity, prior preeclampsia and other pregnancy complications, current pregnancy complications, maternal age below 19 years or above 35 years, and assisted reproduction (Bartsch, Medcalf, Park, & Ray, 2016; Shen et al., 2017; Townsend et al., 2019). Although HDP and other maternal morbidities have decreased globally in the 21st century, the United States has trended in the opposite direction, with worsening maternal health overall and widening disparities among racialized/ethnic groups (MacDorman, Declercq, Cabral, & Morton, 2016; Small, Allen, & Brown, 2017; Tanaka et al., 2007). Racialized and ethnic categories are social and cultural constructs, and higher rates of maternal morbidity and mortality among U.S. Black and American Indian/Alaskan Native women are rooted in structural and cultural racism, disadvantage, discrimination, and resulting inequalities (Boakye et al., 2021; Johnson & Louis, 2022; O'Reilly, 2020; Williams, Lawrence, & Davis, 2019). Structural inequalities (e.g., in access to and experiences with health care, housing, and education) have myriad impacts on SDOHs (e.g., socioeconomic status, health behaviors), and, with the exception of nulliparity, many maternal hypertensive risk factors vary among racialized/ethnic groups owing to inequities affecting health care, behaviors, reproductive decisions, and maternal prepregnancy and gestational conditions (Johnson & Louis, 2022; Williams et al., 2019).

Allostatic load refers to the cumulative psychological and physiological effects of stress experienced across the life course (Guidi, Lucente, Sonino, & Fava, 2021; McEwen & Stellar, 1993), and many of the risk factors associated with HDP (e.g,. overweight, obesity, chronic hypertension) are also associated with maternal stress and allostatic load (Lueth et al., 2022; Walsh et al., 2019). Indices of allostatic load are designed to capture cardiovascular, metabolic, inflammatory, and neuroendocrine profiles by scoring specific biomarkers (e.g., cortisol, cholesterol, and systolic and diastolic blood pressure) that respond to stress (Rodriguez et al., 2019). Stress has been established as a major pathway to racialized/ethnic health disparities (Dunkel Schetter et al., 2013; Louis, Menard, & Gee, 2015; Riggan, Gilbert, & Allyse, 2021), and higher allostatic load scores have been demonstrated to associate with increased risk of HDP (Hux & Roberts, 2015; Lueth et al., 2022). We hypothesize that biomarkers of allostatic load can link upstream SDOHs with downstream HDP to assess pathways leading to disparate morbidity outcomes among different racialized groups in the United States.

Although racialized/ethnic identity is often modeled as a control variable or independent risk factor in multivariate models, this and other demographic variables are insufficient proxies of more complex mechanisms at work and should be appropriately conceptualized in relation to other variables of importance (Clare, 2022; Jeffries et al., 2019; Spector & Brannick, 2011). For example, when racialized/ethnic identity is modeled as an independent covariate, resulting associations with HDP must be inferred as independent of related cumulative social influences on maternal condition. In turn, group-specific risks are interpreted relative to a reference level (e.g., non-Hispanic White women), whereas other significant risk factors identified are inferred to operate equivalently across racialized/ethnic groups. We hypothesize that pathways to HDP may vary in magnitude and salience among racialized groups in the United States owing to sociostructural inequalities and thus use a multigroup modeling framework to estimate group-specific pathways. We developed a SEM using data from the Nulliparous Pregnancy Outcomes Study: Monitoring Mothers-to-be (nuMoM2b) to assess pathways between the social environment, health behaviors, markers of allostatic load, and HDP among non-Hispanic Black and non-Hispanic White first-time mothers in the United States (Haas et al., 2015).

Methods

Study Overview

This is a secondary analysis of data available from the nuMoM2b study (Haas et al., 2015). This observational cohort study was initiated by the Pregnancy and Perinatology Branch of the Eunice Kennedy Shriver National Institute of Child Health and Human Development to address adverse pregnancy outcomes among a diverse sample of U.S. women who had little to no pregnancy history to inform risk interventions. The study was conducted across 17 clinical sites in eight different regions of the United States from 2010 to 2013. All participants provided written informed consent and each site's local institutional review board approved all protocols and procedures (Haas et al., 2015).

A total of 10,037 nulliparous pregnant women were recruited for study participation between 6 and 13 weeks gestation and participated in surveys, interviews, and assessments at four time points across their first trimester, early to mid second trimester, late second to early third trimester, and at birth. Exclusion criteria included maternal age under 13 years, history of three or more spontaneous abortions, evident fetal malformation, known fetal aneuploidy, assisted reproduction, multifetal gestation, or multifetal reduction. Additional details of the original nuMoM2b study design and methods were published by Haas et al. (2015).

The analysis presented in this article was developed for the National Institute of Child Health and Human Development Decoding Maternal Morbidity Data Challenge. The Challenge was launched in 2021 to develop computational solutions that address factors affecting maternal morbidities and disparities in the United States using the nuMoM2b dataset. Participants were required to register with the Challenge and National Institute of Child Health and Human Development Data and Specimens Hub and to comply with the Data and Specimens Hub Data Use Agreement to access the dataset.

Modeling Framework

We developed a SEM using the "lavaan" package in R v.4.1.1 (R Core Team, 2021; Rosseel, 2012). SDOHs were modeled as latent factors to account for measurement error in metrics that are proxies of lived socioecology (Bollen, 2002), and the variables in the nuMoM2b dataset used to characterize the social environment were household income, number of people living in the household, marital status, educational attainment, and partner's educational attainment (Figure 1, Table S1). Latent constructs of behavioral exposures were modeled from measures of physical activity, smoking, and diet. The physical activity factor captures metabolic equivalent of task minutes per week reported across the first three gestational study visits, and the smoking factor reflects daily cigarette usage reported across all four study visits and prior to conception. nuMoM2b study participants reported their dietary intake around the time of conception in a food frequency questionnaire from which alternative healthy eating index scores were derived. To capture a more salient dimension of a poor diet, the dietary factor in our SEM is modeled only on

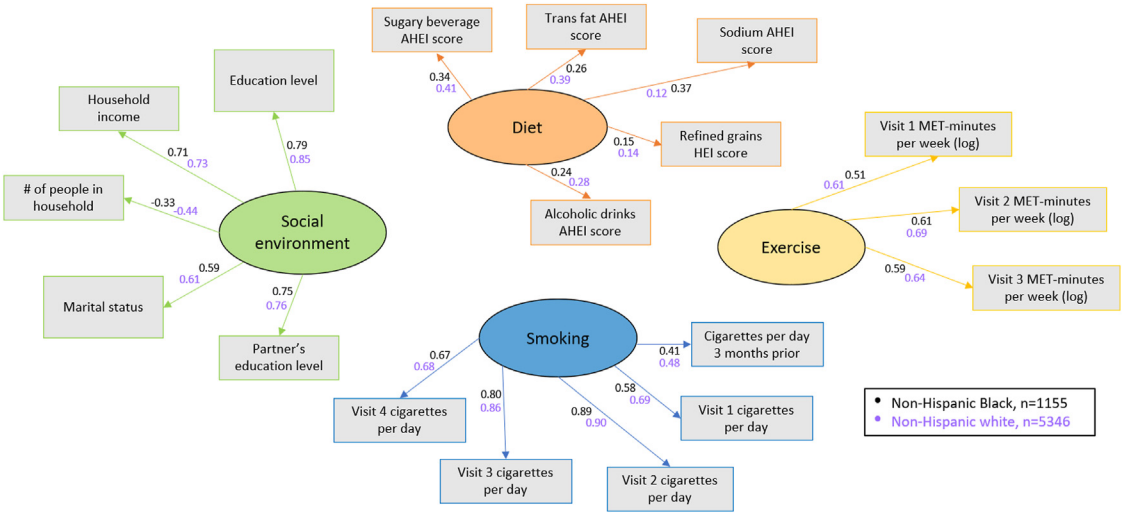

Figure 1. Standardized multigroup factor loadings for social environment, diet, exercise, and smoking latent constructs. AHEI, alternative healthy eating index; MET, metabolic equivalent of task.

alternative healthy eating index scores of refined grains, sodium, sugary beverages, trans fat, and alcoholic drinks (Figure 1).

Allostatic markers in the nuMoM2b dataset included systolic blood pressure, diastolic blood pressure, and weight collected across the four study visits. Using the "lme4" package in R, we fit random intercept and slope models by individual with these longitudinal markers to capture gestational trajectories in changing blood pressure and weight gain (Bates, Mächler, Bolker, & Walker, 2015; R Core Team, 2021). The extracted intercepts and slopes were subsequently included in the SEM to assess clinical hypertensive risk in relation to baseline intercepts during the first trimester of pregnancy as well as the magnitude of sloping changes in these metrics across gestation.

We modeled pathways to five hypertensive outcomes diagnosed in the dataset (see Supplemental Materials for nuMoM2b diagnostic criteria): superimposed preeclampsia, severe preeclampsia, mild preeclampsia, new-onset antepartum hypertension, and new-onset intrapartum or postpartum hypertension. Control variables to account for baseline maternal physiology and confounding influences on gestational hypertensive risk included diagnosis of pregestational diabetes, glucose tolerance test results, reported lifetime number of pregnancies, vaginal bleeding during pregnancy, and any complications in prior pregnancies (defined as the presence of any recorded complications or spontaneous birth or miscarriage before 20 weeks gestation in a previous pregnancy) (Figure 2). Maternal age and whether or not the current pregnancy was planned were modeled as mediators to capture hypertensive effects of the social environment that operate through fertility decisions (Nelson et al., 2022; Umesawa & Kobashi, 2017). Although hypertensive risks generally increase with maternal age, which may in part reflect cumulative impacts of chronic social stressors, higher levels of education and income are also associated with later age at first birth and planned pregnancy (Schummers et al., 2018; Sheppard, Pearce, & Sear, 2015), which may mediate the associations among these factors in first-time mothers. Diagnosed chronic hypertension was included as a moderator to blood pressure and weight trajectory intercepts to

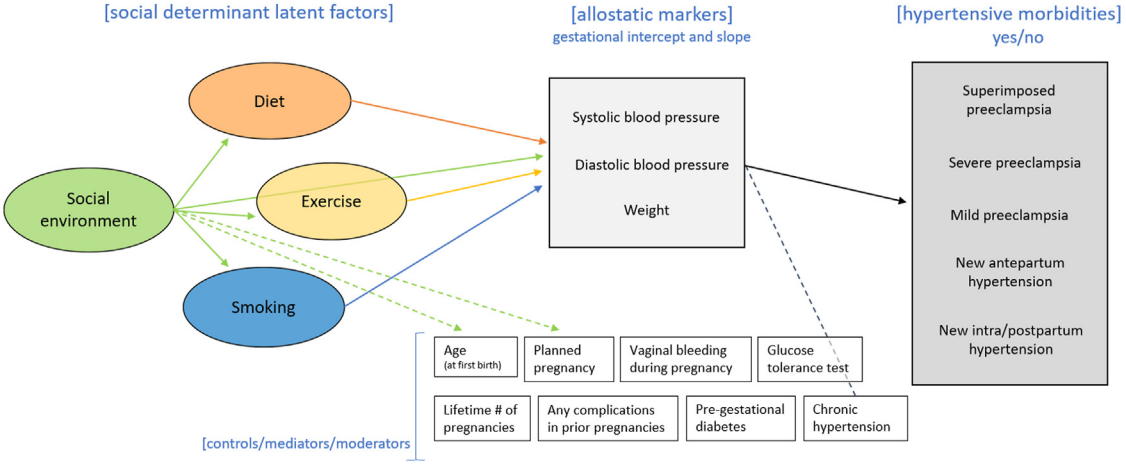

Figure 2. Structural equation model path diagram between latent social determinants of health, allostatic marker baselines and gestational trajectories, and hypertensive pregnancy outcomes.

account for the elevated baseline blood pressure and weight levels early in pregnancy among these individuals and as a distinguishing predictor between superimposed preeclampsia and other hypertensive outcomes.

We fit this SEM in a multigroup framework for non-Hispanic Black and non-Hispanic White identities, hypothesizing that the pathways by which SDOHs become embodied as disparate hypertensive risks may vary in salience and magnitude across groups owing to experienced discrimination, perceived stress, and other factors associated with sociostructural inequities (Walker, Strom Williams, & Egede, 2016). Furthermore, metrics such as income and educational attainment used to proxy socioeconomic environments may not capture comparable circumstances given that there are often significant disparities in loan debt, household assets, and other aspects of generational wealth between White Americans and other groups (Houle & Addo, 2019). We also ran this SEM for a collapsed grouping of women in other minoritized groups (Asian, Hispanic, Multiracial, Native American/Hawaiian, and other identities). Owing to small sample sizes, we were unable to examine group-specific pathways among these identities but have included the results from this heterogeneous sample in the Supplemental Material (Tables S1–S3). After excluding individuals with undocumented hypertensive pregnancy outcomes, the sample available for this SEM consisted of 1,155 non-Hispanic Black and 5,346 non-Hispanic White women for a combined total of 6,501 first-time mothers in these two racialized groups (Table 1).

Results

Sample Statistics and Maternal Health Vary by Racialized Identity

Thirty-two percent of Black women (370/1,155) in this sample of nuMoM2b participants developed a hypertensive morbidity during or resulting from pregnancy, compared with 23% of White women (1,235/5,346) and 23% of women across all racialized/ethnic groups (2,040/8,772) in these data (Table 1, Table S2). The most severe morbidities of superimposed and severe preeclampsia show the greatest disparities, and Black women have higher morbidity rates than all other groups across all hypertensive conditions with the exception of new-onset antepartum hypertension (Table S2). Black women in this sample also have rates of diagnosed chronic hypertension and pregestational diabetes more than 2.5 times higher than those of other minoritized groups and non-Hispanic White women. Additionally, Black mothers-to-be were 2.9 times less likely than White women to have planned on becoming pregnant and had marriage rates 5.1 times lower than White women in this sample (Table 1). The average annual household income among Black mothers-to-be is less than $25,000, compared with approximately $45,000 among other minoritized women and more than $60,000 among White women (Table S2).

Latent Constructs of Social and Behavioral Determinants

We confirmed the SDOH latent factors of the model in a tandem multigroup manner and derived group-specific factor loadings of social environment, diet, exercise, and smoking factors on their indicator variables (Figure 1, Table S2). Fit statistics indicate good support for this latent framework (comparative fit index [CFI] = 0.940; root mean square error of approximation = 0.045; root mean square residual = 0.052), and smoking and social environment factors show the strongest relationships with their indicators. The dietary construct has the weakest standardized loadings, but sugary beverage and sodium scores correlate to the greatest extent. Educational attainment and household income load most strongly onto the social environment construct. Education metrics show larger effects among non-Hispanic White women, whereas annual household income impacts this factor to the greatest magnitude among Black women (Figure 1, Table S2).

Allostatic Pathways Link Social Determinants With Hypertensive Pregnancy Outcomes

The final sample included in the SEM had 999 Black women and 4,689 White women because the modeling framework can accommodate some, but not all, patterns of missingness in the data. The full SEM shows good fit among both racialized groups ($CFI_{Black} = 0.904$, $CFI_{white} = 0.905$), and many of the statistically significant risk pathways to HDP vary among groups as well (Figure 3, Table S3). Standardized path coefficients reflect the influence of each predictor relative to the others (measured on varying scales) on the mean outcome response, whereas unstandardized coefficients estimate the linear change in response per unit change in each predictor. Figure 3 shows only standardized path coefficients that are statistically significant at the 0.05 level to facilitate group comparisons of the most impactful hypertensive risk factors in these data (Table S3).

Aspects of the social environment are the most salient predictor of hypertensive morbidities in this nuMoM2b sample, with effects that vary by racialized identity (Figure 3, Table S3). The social environment shows impacts on early pregnancy baselines and gestational trajectories of blood pressure and weight both directly and indirectly via effects on dietary, exercise, and smoking behaviors. Higher (more supportive) social environment scores associate with relatively reduced gestational weight gain but greater baseline weight. Out of the allostatic markers modeled here, baseline systolic blood pressure and gestational weight gain occur most frequently in statistically significant risk paths to hypertensive outcomes (Figure 3).

Hypertensive Risk Pathways Vary Among Racialized Groups

The nine largest path coefficients are concentrated among Black women, and five of these paths are rooted in the social environment latent factor (Figure 3, Table S3). The effects of the social environment are smaller in magnitude (unstandardized coefficients) on mediating behavioral pathways, and greater in magnitude and salience (standardized coefficients) on direct paths to allostatic markers among Black women as compared to other racialized/ethnic groups (Figure 3, Table S3). For example, although every unit score increase in the dietary factor (healthier diet) decreases baseline weight by a predicted 55 pounds among Black women and by 16 pounds among White women, the direct effects of the social environment on baseline weight are relatively more impactful than dietary scores among Black women. Relative to the average weight gain in Black women of 9.5 pounds each trimester, a one score increase in the social environment corresponds with a decrease in weight gain by an average of 11.5 pounds each trimester, compared with a decrease of 0.7 pounds among White women in this sample (Table S3). Conversely, the mediating effect of the social environment on dietary scores is more than twice as large among White women (and among other minoritized women) as compared with Black women (Figure 3, Table S3).

Table 1
Descriptive Sample Statistics Grouped by Non-Hispanic Black and Non-Hispanic White Identities in the Nulliparous Pregnancy Outcomes Study: Monitoring Mothers-to-Be Data

Variable Counts	Non-Hispanic Black $n = 1155$	Non-Hispanic White $n = 5346$
Superimposed preeclampsia	20 (1.7)	24 (0.5)
Severe preeclampsia	56 (4.9)	136 (2.5)
Mild preeclampsia	50 (4.3)	126 (2.4)
New-onset antepartum hypertension	78 (6.8)	423 (7.9)
New-onset intra/postpartum hypertension	166 (14.4)	526 (9.8)
Diagnosed chronic hypertension	67 (5.8)	114 (2.1)
Pregestational diabetes	40 (3.5)	66 (1.2)
Lifetime no. of pregnancies	1.46 (1.00, 7.00)	1.29 (1.00, 6.00)
Any complications in prior pregnancies	187 (16.2)	850 (15.9)
Vaginal bleeding during current pregnancy	81 (7.0)	333 (6.2)
Glucose tolerance test (mg/dL)	103.8 (41.00, 380.00)	110.7 (11.60, 620.00)
Current pregnancy planned	279 (24.2)	3781 (70.7)
Age (years)	23.32 (13.00, 45.00)	28.18 (15.00, 44.00)
Household income level*	5.89 (1.00, 14.00)	10.15 (1.00, 14.00)
No. in household	3.03 (1.00, 13.00)	2.36 (1.00, 17.00)
Married	171 (14.8)	4069 (76.1)
Education level†	2.87 (1.00, 6.00)	4.51 (1.00, 6.00)
Partner's education level†	3.60 (2.00, 8.00)	5.30 (1.00, 8.00)
Sugary beverage AHEI score‡	1.00 (0.00, 10.00)	3.83 (0.00, 10.00)
Trans fat AHEI score‡	7.58 (1.60, 10.00)	8.22 (0.29, 10.00)
Sodium AHEI score‡	4.68 (0.00, 10.00)	6.22 (0.00, 10.00)
Alcoholic drinks AHEI score‡	4.17 (0.00, 10.00)	5.34 (0.00, 10.00)
Refined grains HEI score‡	7.82 (0.00, 10.00)	7.97 (0.00, 10.00)
Visit 1 MET-minutes per week§	643.00 (0.00, 4040.00)	929.4 (0.00, 4524.00)
Visit 2 MET-minutes per week§	624.00 (0.00, 4880.00)	812.8 (0.00, 5435.00)
Visit 3 MET-minutes per week§	526.90 (0.00, 4176.00)	720.9 (0.00, 3820.00)
3 Months prepregnancy cigarettes per day	4.13 (0.00, 40.00)	3.35 (0.00, 60.00)
Visit 1 cigarettes per day	0.72 (0.00, 20.00)	0.71 (0.00, 40.00)
Visit 2 cigarettes per day	0.25 (0.00, 10.00)	0.31 (0.00, 25.00)
Visit 3 cigarettes per day	0.29 (0.00, 20.00)	0.29 (0.00, 50.00)
Cigarettes per day at delivery	0.21 (0.00, 20.00)	0.29 (0.00, 70.00)
Visit 1 systolic blood pressure (mm Hg)	111.80 (79.00, 165.00)	109.5 (66.00, 168.00)
Visit 2 systolic blood pressure (mm Hg)	111.20 (75.00, 169.00)	110.20 (72.00, 165.00)
Visit 3 systolic blood pressure (mm Hg)	111.80 (72.00, 161.00)	111.10 (78.00, 170.00)
Visit 1 diastolic blood pressure (mm Hg)	67.77 (30.00, 111.00)	67.23 (40.00, 108.00)
Visit 2 diastolic blood pressure (mm Hg)	66.00 (42.00, 100.00)	66.74 (40.00, 115.00)
Visit 3 diastolic blood pressure (mm Hg)	66.61 (40.00, 128.00)	67.41 (40.00, 170.00)
Visit 1 weight (lbs)	172.90 (82.80, 368.00)	155.70 (73.00, 448.00)
Visit 2 weight (lbs)	179.00 (87.50, 365.00)	161.50 (77.20, 344.00)
Visit 3 weight (lbs)	187.30 (93.0, 387.00)	172.40 (83.60, 353.00)
Weight before giving birth (lbs)	201.00 (106.00, 457.00)	187.00 (70.00, 389.00)

Abbreviation: AHEI, alternative healthy eating index.
Note: Values are number (%), or numeric = mean (min, max).
* Household income levels: 1 = <$5,000; 2 = $5,000–9,000; 3 = $10,000–15,000; 4 = $15,000–20,000; 5 = $20,000–25,000; 6 = $25,000–30,000; 7 = $30,000–40,000; 8 = $40,000–50,000; 9 = $50,000–60,000; 10 = $60,000–75,000; 11 = $75,000–100,000; 12 = $100,000–150,000; 13 = $15,0000–200,000; 14 = $200,000 or more.
† Education levels: 1 = 8th grade or less; 2 = 9th–12th grade no diploma; 3 = high school or GED completed; 4 = some college no degree; 5 = associate/technical degree; 6 = Bachelor's degree; 7 = Master's degree; 8 = Doctorate or professional degree.
‡ Health eating index scores: range 0–10, higher scores indicate closer conformance to recommended dietary patterns.
§ MET-minute: metabolic equivalent (kcal/kg) of energy expended per minute compared with being at rest.

Each social environment unit increases systolic blood pressure rise by an average of 2.6 mm Hg and increases diastolic rise by an average 1.0 mm Hg among Black women across each trimester but shows no significant impacts on blood pressure slopes among any other racialized/ethnic group (Figure 3, Table S3). Higher diet scores predict lower systolic (−3.3 mm Hg/trimester) and diastolic (−2.3 mm Hg/trimester) blood pressure trajectories among Black women and are not significantly predictive of these slopes among other racialized groups in this sample. Gestational weight gain is the most frequently significant predictor of HDP among Black mothers-to-be, increasing risks of superimposed preeclampsia by 3%, mild preeclampsia by 5%, and new-onset antepartum hypertension by 6% for every additional 10 pounds gained beyond the group average of 9.5 pounds each trimester (Figure 3, Table S3).

Several of the control and mediator variables in this SEM also show disparate associations and hypertensive risks among racialized groups. Although the social environment is more strongly associated with higher age at first birth in Black women as compared to other racialized/ethnic groups, higher age is only significantly predictive of new intra/postpartum hypertension (0.3% increased risk per year) among White women in this sample (Figure 3, Table S3). Diagnosed pregestational diabetes associates with increased hypertensive risk in all groups, but

with more severe morbidity outcomes in Black women (17.8% increased risk of superimposed preeclampsia) than among White women (12.1% increased risk of new-onset antepartum hypertension).

Discussion

HDPs show disparate incidence across racialized/ethnic groups among this national sample of nulliparous U.S. women (Table 1, Table S2). Relative to White women, Black mothers-to-be had increased odds of HDP (unadjusted odds ratio, 1.39), and these disparities are consistent with other estimates from U.S. data that have been fundamentally attributed to impacts of structural racism, cultural racism, and individual experiences of discrimination among Black Americans (Burger et al., 2022; Johnson & Louis, 2022; Minhas et al., 2021; Williams et al., 2019). These nuMoM2b data also show that nulliparous Black women were three times as likely to have been diagnosed with chronic hypertension than women in White or other minoritized groups (Table 1, Table S2), increasing their risk of superimposed preeclampsia by 20.0% (Table S3) and demonstrating that disparities in prepregnancy health directly impact maternal morbidity and birth outcomes.

In our model, disparities in household income and educational attainment show the strongest loadings onto the social environment latent factor, and this factor has the most significant impacts on hypertensive pathways out of all included predictors (Figures 1 and 3, Table S1). These factor and pathway findings support previous analysis of the nuMoM2b dataset demonstrating that women with the lowest household incomes and highest reported stress have the highest overall risk of maternal morbidity (Erickson & Carlson, 2022). Although high levels of income and education are known to associate with and mediate maternal morbidity risks in the United States (Howland et al., 2019), they do not confer equitable benefits across racialized groups (Love, David, Rankin, & Collins, 2010; O'Campo et al., 2016). Higher levels of socioeconomic status show diminishing returns among Black women as compared with White women in attenuating risks of HDP and adverse birth outcomes (Miller et al., 2020; Ross et al., 2019), and Black Americans are even more likely to experience chronic and acute discrimination with greater upward economic mobility (Colen, Ramey, Cooksey, & Williams, 2018). This factor, coupled with evidence of higher stress among workers with greater socioeconomic status observed elsewhere (Damaske, Zawadzki, & Smyth, 2016), may partially account for our finding that higher social environment scores associated with higher baseline weight and increased blood pressure measures, with stronger effects in Black women along both pathways (Figure 3, Table S3). In other words, socioeconomic status may differently influence stressors affecting allostatic load prior to and during pregnancy, with effects compounded by racialized experiences among Black women.

The construct of allostatic load was initially developed to measure the physiological impacts of cumulative stress and environmental challenges on the body that predispose individuals to more severe morbidities (McEwen & Stellar, 1993), and these measures seem to capture a significant dimension of racialized health disparities beyond that attributed to the linear effects of SDOHs variables (O'Campo et al., 2016; Riggan et al., 2021). Our SEM included three allostatic markers (systolic and diastolic blood pressure, gestational weight gain) measured longitudinally across gestation and demonstrated good fits in linking these clinical measures with SDOHs and HDP in a multigroup framework. Pregnancy induces a unique form of cardiac hypertrophy and physiological stress (Chung & Leinwand, 2014), which warrants specific consideration when assessing clinical predictors of HDP. Our model results support other analyses demonstrating that elevated blood pressure early in pregnancy as well as increasingly sloped blood pressure trajectories across gestation are both predictive of HDP (Hauspurg et al., 2019; Hermida et al., 2000). The unique physiological stressors of pregnancy impact allostasis broadly, and higher allostatic load early in pregnancy is known to predict preeclampsia and adverse perinatal outcomes, although few studies have assessed trajectories of allostatic load during pregnancy beyond the first trimester (Li, Dalton, Lee, Rosemberg, & Seng, 2020; Lueth et al., 2022). Gestational trajectories of allostatic markers should be considered independently from and in

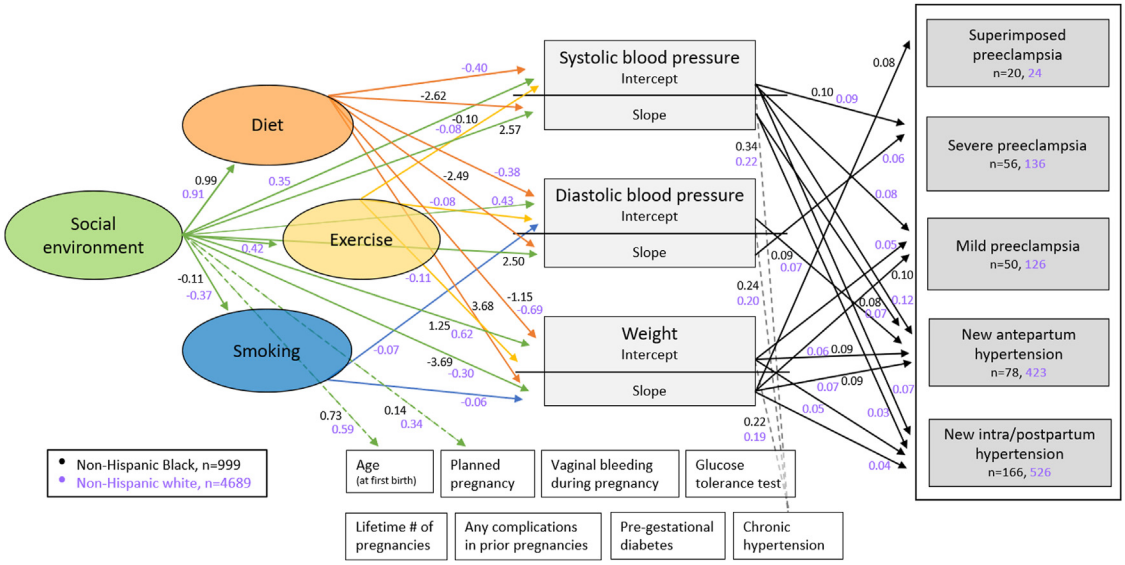

Figure 3. Standardized path coefficients from the structural equation model significant at the 0.05 level among non-Hispanic Black and non-Hispanic White groups.

addition to clinical thresholds of cross-sectional measures, and more work is needed to assess biomarkers comprehensively across gestation to enhance screening for high-risk outcomes and maternal morbidities (Hermida et al., 2000; Vianna, Bauer, Dornfeld, & Chies, 2011).

Good SEM fit statistics support our hypothesis that embodied stress captured in metrics of allostatic load can link upstream SDOHs with disparities in downstream maternal morbidity (Table S3). Another analysis of these nuMoM2b data published by Lueth et al. (2022) also found that high allostatic load scores during the first trimester of pregnancy predicted diagnosis of HDP, and allostatic load was a statistically significant mediator between racialized identity and hypertensive outcomes in multivariable logistic regression. Lueth et al. (2022) specifically found weaker associations between allostatic load and HDP among non-Hispanic Black women as compared with non-Hispanic White women in this sample. Our SEM identified fewer significant pathways between allostatic markers and HDP among the non-Hispanic Black group, but coefficients were not smaller in magnitude than those estimated among non-Hispanic White women (Table S3). Together, these analyses highlight the role of embodied stress as a major pathway to maternal morbidity and demonstrate the importance of structuring models to account for racialized disparities that mediate other associations.

Implications for Policy and/or Practice

This analysis underscores that sociostructural inequalities are more influential than individual behaviors in perpetuating racialized health disparities and that embodied stress is a major pathway to disparate maternal morbidity outcomes. Although behavioral recommendations (e.g., dietary and exercise modifications, meditation) remain primary tools for mitigating physiological impacts of stress (Centers for Disease Control and Prevention, 2022; The Lancet Editorial, 2022), policies targeting systemic and structural stressors beyond the individual level are needed to address the upstream causes (APA, 2022; Churchwell et al., 2020). Disparities in sociodemographic metrics and their mediating effects demonstrate the multidimensionality of SDOHs along causal pathways, and policies aiming to reduce disparities by targeting SDOHs must be conceptualized in a multi-sectoral framework that considers the variable contexts of specific factors among racialized groups (Chantarat, Van Riper, & Hardeman, 2022).

Additional work is needed to incorporate more robust measures of SDOHs beyond those captured in basic demographic profiles to identify the most promising avenues for policy and intervention (Elias, Jutte, & Moore, 2019; Johnson & Louis, 2022). Comprehensive assessments of the many environmental, structural, and behavioral dimensions of SDOHs are challenging to aggregate in practice (Jeffries et al., 2019), and while measuring embodied impacts of racism and discrimination is critical to improving health equity, quantifying structural racism and its health effects remains rare (Brown & Homan, 2022).

Limitations

The nuMoM2b data were collected from a nationally diverse sample of U.S. women, but as participants were enrolled at university affiliated hospitals during their first trimester (Haas et al., 2015), the sample may not represent women with more limited access to medical and prenatal care (Krukowski et al., 2022), nor the growing proportions of women giving birth at home or in birth centers (MacDorman & Declercq, 2019). Considering that all study sites were located in metropolitan areas, the extent to which these data capture rural health disparities is also limited. 22.8% of U.S. women lived rurally in 2010 (Fields, Holder, & Burd, 2016), and rural women show higher rates of both prepregnancy hypertension and HDP, with racialized disparities that persist across rural and urban areas (Cameron et al., 2020, 2022). Additionally, we were not able to examine varied HDP risk pathways for women who identified as Hispanic, Asian/Pacific Islander, Multiracial, Native American/Hawaiian, or Other in these data owing to small group sample sizes.

Last, blood pressure thresholds for clinical diagnosis of general hypertension were lowered in 2017 based on their associations to cardiovascular morbidities among non-pregnant adults (Whelton et al., 2018), and pregnant women show elevated risk for severe maternal morbidities at these lowered thresholds as well (Hauspurg et al., 2019). These data from 2010 to 2013 used hypertensive thresholds of systolic blood pressure of 140 mm Hg or higher and diastolic blood pressure of 90 mm Hg or higher. Applying lower cutoffs in future assessments would identify more women at risk of developing HDP and may identify additional risk factors by reclassifying women with blood pressures in elevated (120–129 mm Hg systolic/<80 mm Hg diastolic) and stage 1 (130–139 mm Hg systolic/80–89 mm Hg diastolic) hypertensive ranges.

Conclusions

This analysis demonstrates the usefulness of pathway modeling with large datasets, presents a framework that can address racialized/ethnic health disparities by estimating group-specific risk pathways, and can be expanded to additional health predictors and disparities in the future. Estimates from this SEM show that social inequalities are the most impactful predictors of HDP among a national sample of nulliparous U.S. women, and disparities in prepregnancy health also lead to more severe morbidity outcomes among Black women. For Black mothers in particular, the social environment shows stronger direct effects on allostatic markers than via behaviorally mediated dietary, exercise, or smoking paths. Pathways through baseline levels and gestational trajectories of allostatic markers underscore that embodied stress is a major path driving maternal health disparities in the United States.

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Supplementary Data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.whi.2023.08.001.

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Author Descriptions

Monica H. Keith, PhD, is an Assistant Professor in the Department of Anthropology at Vanderbilt University. She earned her PhD from the University of Missouri in 2020 and researches disparities in longitudinal health, child growth, and reproductive outcomes.

Melanie A. Martin, PhD, is an Assistant Professor in the Department of Anthropology at the University of Washington. She earned her PhD from the University of California Santa Barbara in 2015 and researches biocultural influences on growth, development, and reproduction.