Pregnancy as a Window to Racial Disparities in Hypertension

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Abstract

Background: The black-white disparity in hypertension (HTN) among U.S. women persists after accounting for known risk factors. Pregnancy complications may reveal increased risks for later HTN. We examined the contribution of HTN risk factors measured at both midlife and pregnancy to black-white disparities in midlife HTN.

Methods: Data came from a Michigan-based longitudinal study beginning in pregnancy. At 7–15 years post-pregnancy ($n=615$, mean age=37), women were assessed for cardiovascular health, including blood pressure, and categorized as hypertensive ($n=126$), prehypertensive ($n=149$), and normotensive ($n=340$). Midlife risk factors for HTN were assessed in four domains: socioeconomic status (SES), psychosocial, behavioral, and physiological. We used generalized logit models to assess the degree to which each domain attenuated the black (vs. white) odds ratio (OR) for HTN at midlife. We then added indicators of pregnancy health, that is, preterm delivery, prepregnancy body mass index (BMI), C-reactive protein (CRP) levels, depressive symptoms, smoking, hypertensive disorders, and lipid levels.

Results: Black women had 3.3 (95% CI: 2.0–5.5) times the odds of HTN compared to white women after adjusting for age. Following adjustment for midlife SES, and psychosocial, behavioral, and physiological factors, the OR was 2.1 (95% CI: 1.2–4.0). Adjustment for prepregnancy BMI, CRP, and depressive symptoms during pregnancy reduced the OR to 1.9 (95% CI: 1.0–3.7).

Conclusions: Known risk factors measured at midlife explained some, but not all, of the race disparity in midlife HTN. Indicators of pregnancy health also contributed to the race disparity in HTN at midlife.

Keywords: hypertension, pregnancy, women, African Americans, blood pressure

Introduction

BLACK WOMEN in the United States have rates of hypertension (HTN) 50% higher than white women¹ and are 40% more likely to develop cardiovascular disease (CVD),² for which HTN is a key risk factor.³ HTN is a particularly important CVD risk factor for both women and blacks, for whom the percent of CVD in the population attributable to HTN is 50% and 100% higher compared to men and whites, respectively.⁴

Although the black-white disparity in HTN may be, in part, attributable to differences in obesity—particularly abdominal obesity—vitamin D status, socioeconomic status (SES), and the neighborhood environment,^{5–10} the majority of studies report that disparities persist even after adjustment for a large

set of risk factors.^{5,6,9,11} Importantly, despite the noted importance of investigating sex-specific determinants of cardiovascular health,² little research has focused on understanding the determinants of the black-white disparity in HTN among women. Moreover, most studies do not include factors measured before the onset of HTN, which could help identify early targets for prevention or intervention.

Pregnancy may provide a unique window into cardiovascular risk^{12,13} and black-white disparities in cardiovascular health. For example, women with preeclampsia exhibit two-fold increased risks of CVD later in life and a 270% increase in risk of HTN (compared to women with uncomplicated pregnancies).¹⁴ Delivering preterm (vs. term)—which is 50% more common among black compared to white women¹⁵—has also been linked with later-life risk of CVD¹⁶ and with

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Presented as a moderated poster session at the American Heart Association EpiLifestyles Scientific Sessions in Portland, Oregon in March 2017.

more rapid increases in blood pressure over 20 years following pregnancy.¹⁷ Even moderately elevated blood pressure (vs. normal blood pressure) during pregnancy is associated with over twice the odds of HTN in later life.¹⁸ Thus, pregnancy could provide an opportunity to identify factors contributing to later-life disparities in women's CV health. As the vast majority of pregnant women in the United States receive at least one prenatal care visit, pregnancy may be a particularly relevant opportunity to mitigate race disparities in long-term health.

To our knowledge, no existing research has examined the contribution of risk factors measured during pregnancy as well as later in life to black-white disparities in HTN among women. Using a unique cohort of black and white women enrolled during pregnancy and followed up 7 to 15 years later (mean age = 37, range 25–58, hereafter “midlife”), we examined the degree to which (1) known HTN risk factors measured at midlife explain the black-white disparity in midlife HTN and (2) markers of pregnancy health explain the black-white disparity in midlife HTN.

Methods

Data and study population

We used data from the POUCHMoms study (2011–2015), a one-time follow-up study that assessed cardiovascular health 7 to 15 years after delivery (“midlife”) among a subset of women enrolled in a pregnancy cohort (POUCH [Pregnancy Outcomes and Community Health] Study, 1998–2004). The POUCH Study enrolled 3,019 women in the 16th–27th week of a singleton pregnancy at 52 clinics in 5 Michigan communities. A detailed description of the POUCH Study has been published previously.¹⁹ At enrollment, all women were interviewed and biological samples were collected. A case-cohort design was used to create a subcohort ($n = 1,371$) of all women delivering preterm and a stratified sample of all term births. POUCH Study participants had similar sociodemographic characteristics when compared to the general population of mothers giving birth in the five communities, although POUCH included a smaller percentage of black women older than 30 years than in the communities.¹⁹

The POUCHMoms Study invited all women from the subcohort to participate in a follow-up visit at 7–15 years postpartum. A trained research nurse carried out in-person interviews and self-administered questionnaires, biological sample collection, and measurement of height, weight, and blood pressure. Of the 1,371 eligible women, 224 declined, 12 were deceased since last follow-up, 41 were ineligible due to current pregnancy or living out of state, 26 did not attend a scheduled follow-up appointment, and 390 could not be located. POUCHMoms thus enrolled 678 women, and our analytic sample included 615 non-Hispanic black and white women without prepregnancy HTN.

Conceptual framework

Figure 1 illustrates the hypothesized relationships among race, risk factors, and HTN. Race is associated with SES, a distal determinant of HTN; both race and SES influence psychosocial and behavioral factors. Psychosocial and behavioral factors can impact each other (e.g., sleep may affect mental health and vice versa) and also affect more proximal, physiological determinants of HTN such as body mass index (BMI). Risk factors depicted in Figure 1 may act at one or more time points or accumulate across the life course to affect HTN. Our study seeks to determine whether including such factors measured at pregnancy helps explain the race disparity in HTN later in life above and beyond including these risk factors measured concurrently with later HTN.

Measures

Our primary outcome was HTN at midlife. Blood pressure was measured by the study nurse thrice on the participants' dominant arm at 1-minute intervals following a 5-minute seated rest, and an average of the second and third measurement was used to determine hypertensive status. We categorized women as hypertensive (systolic BP of ≥ 140 or a diastolic BP of ≥ 90 , or self-reported use of antihypertensive medications), prehypertensive (systolic BP of 120–139 or a diastolic BP of 80–89, and not hypertensive), or normotensive. In secondary analyses, we also examined continuous measures of systolic blood pressure, diastolic blood pressure,

FIG. 1. Hypothesized risk factor pathways—from most distal to most proximate—through which race may affect hypertension.

and the average of systolic and diastolic blood pressure, which has been shown to be the single measure of blood pressure most informative for predicting mortality.²⁰ Race was categorized based on self-reported race/ethnicity at the time of pregnancy as non-Hispanic black or white (hereafter “black” and “white”).

HTN risk factors at midlife included age as well as variables from the four categories of established risk factors depicted in Figure 1: SES and psychosocial factors, behavioral factors, and physiological factors.

Midlife SES. We assessed SES using a summary measure (based on previous work using the POUCH data²¹), whereby women received one point for each of the following indicators: >12 years of education (vs. ≤12 years), married (vs. unmarried), income >\$50,000/year (vs. ≤\$50,000/year), reporting having any investments (vs. no investments), and reporting having private health insurance (vs. Medicaid or no insurance). Points were summed and categorized as low (<25th percentile), moderate (25th–75th percentile), or high (>75th percentile). We also tested models using each individual measure of SES (e.g., education and marital status).

Midlife psychosocial factors. Psychosocial factors included depression, hostility, and job strain, all of which have been shown to be associated with cardiovascular health in prior studies.^{22,23} Depression was scored based on the application of the DSM-V criteria to women’s responses to the Composite International Diagnostic Interview (CIDI) depression module and was categorized as follows: no depression, major depression (women who met all DSM-V criteria, including episode duration of at least 14 days), and short duration symptomatology (women who met all DSM-V criteria except episode duration, which ranged from 3 to 13 days). Hostility, a continuous variable, was based on the summary of a woman’s responses to the Cook-Medley scale.²⁴ Job strain was scored based on women’s responses to a modified version of the Karasek demand-control questionnaire.²⁵ Responses to questions related to both job psychological demand and decision latitude (see Appendix Table A1 for questions) were summed and divided into “low” (below the median) and “high (above the median). Women were then categorized as having low-strain (low demands and high decision), active (high demands and high decision), passive (low demands and low decision), or high-strain (high demands and low decision) jobs.

Midlife behavioral factors. Behavioral factors were all self-reported and included current smoking (yes/no), diet quality score (based on the Block Food Frequency Questionnaire; range 26–90 with higher values representing higher quality diet), number of sedentary hours per week (leisure and work combined), and a summary sleep score (based on the Pittsburgh Sleep Quality Index; range 2–20 with lower values representing better sleep).

Midlife physiological factors. Physiological factors included BMI, blood lipid levels, and blood c-reactive protein (CRP) levels. BMI (kg/m²) was calculated based on measured height and weight. Lipids (high-density lipoprotein cholesterol [HDL-C], low-density lipoprotein cholesterol [LDL-C], and triglycerides) were assayed in blood samples

collected following an overnight fast, and measured at the Heinz Nutrition Laboratory at the University of Pittsburgh using standardized methods,^{26–28} and log-transformed for multivariate analyses. High-sensitivity CRP was measured by ELISA. Two different assay kits were used to measure CRP over the course of the study; thus, we created kit-specific z-scores for analysis of CRP. We also tested models using waist circumference in inches (recorded as the average of three measurements) as a measure of abdominal adiposity instead of BMI.

Pregnancy variables. HTN risk factors measured at pregnancy included SES, BMI, maternal blood lipids and CRP levels (assessed between 16–27 weeks’ gestation), and pregnancy complications (abstracted from the medical record and/or collected at the time of delivery). The SES summary score for pregnancy was based on the woman’s own and the baby’s father’s education and occupation, Medicaid status, and household income.²¹ Prepregnancy BMI was calculated based on self-reported prepregnancy weight and height. HDL-C, LDL-C, triglycerides, and CRP were measured in nonfasting blood samples and analyzed using the same methods as described above. Pregnancy complications included hypertensive disorders of pregnancy (gestational HTN or preeclampsia) and preterm delivery (PTD, <37 weeks). We also calculated BMI change from prepregnancy to midlife.

Statistical analysis

We first calculated the frequency (percent) or mean (standard deviation [SD]) for all outcome measures and risk factors overall and by race, using weighted regression models to detect significant differences by race. We then conducted sequential multinomial logistic regression models for the 3-level outcome (hypertensive, prehypertensive, and normotensive [reference]), adding one category of variables in each step. We entered midlife risk factors into our models in the sequence depicted in Figure 1 (from most distal to most proximate): that is, SES, psychosocial, behavioral, and physiological. We then entered pregnancy variables into the model.

In Model 1, we included only race and age; we consider the odds ratio (OR) for HTN comparing black to white women to quantify the disparity in HTN. As we add covariates to the model, this OR for race represents the disparity that would remain between black and white women if the distributions of other covariates in the black population were set equal to the distributions in the white population.²⁹ Models 2–6 sequentially added variables measured at midlife follow-up, that is, SES summary score, psychosocial factors, behavioral variables, and biological variables. Model 7 added pregnancy variables, and Model 8 substituted BMI change from prepregnancy to midlife for the two separate BMI measures. Before adding variables into models 3–7, we tested each variable within the category (e.g., for psychosocial, we tested depression alone, hostility alone, job strain alone, and every possible combination) in separate age-adjusted regression models to determine which variable or combination of variables from each category decreased the race disparity OR the most.

We used the same approach to conduct sequential linear regression models of average blood pressure. We considered additionally adjusting these models for self-reported use of

antihypertensive medications; however, analyses indicated that the use of antihypertensive medication was positively associated with blood pressure, suggesting that medication serves as a proxy marker of higher blood pressure (instead of reducing blood pressure). Within these linear models, we also tested interactions between race and midlife BMI and between race and age using a Wald test with p -value <0.10 to identify significant interactions. Some of the covariates included in our fully adjusted model with pregnancy variables (Model 6) had missing values: midlife hostility ($n=27$), midlife diet score ($n=1$), midlife CRP z-score ($n=2$), pregnancy depressive symptoms ($n=2$), and pregnancy CRP ($n=28$). We thus re-ran all models using only the complete case sample ($n=562$). Analyses were conducted using SAS 9.4 (Cary, NC) and weighted for the subcohort sampling strata using survey procedures in SAS.

Results

Table 1 displays the frequency (percent) and mean (SD) for all variables overall and by race/ethnicity; we reported the weighted percent and mean for all variables. Our sample included 242 black and 373 white women, and the mean age was 37.7 years. Overall, 18.6% of women were hypertensive and 24.1% were prehypertensive. Compared to white women, black women were significantly more likely to be either hypertensive (28.5% vs. 15.4%) or prehypertensive (26.4% vs. 23.3%) and had higher mean values for systolic, diastolic, and average blood pressure. At 7–15 years after the POUCH Study birth, black women were also significantly more likely to be of low SES (57.6% vs. 19.3%), had higher mean scores for hostility, sedentary time, and BMI, and had lower diet quality scores. Black women were younger and had lower levels of triglycerides compared to white women. There were no statistically significant differences by race in depression, job strain, current smoking, sleep score, HDL-C or LDL-C cholesterol levels, or CRP z-score levels. At pregnancy, black women were also more likely to be of low SES (48.4% vs. 17.0%), to have had a PTD (14.3% vs. 9.5%), and had higher scores on hostility and depressive symptoms, compared to white women. However, black women had healthier lipid levels, lower prevalence of hypertensive disorders of pregnancy, and were not significantly different from white women in terms of prepregnancy BMI or CRP levels during pregnancy.

Our initial investigation of the variables in each domain found that the summary SES score was more strongly associated with HTN and reduced the black-white disparity (OR) more than the individual measures. Within the other domains, the factors that were associated with HTN and reduced the black-white OR when added into the model were as follows: psychosocial—hostility; behavioral—the combination of diet quality score and sedentary time; physiological—the combination of BMI and CRP z-score; and pregnancy—pregnancy BMI, CRP levels, and depressive symptoms (data available upon request). Waist circumference was more strongly associated with HTN than BMI, but contributed less to the black-white disparity; thus we included BMI and not waist circumference in the final models.

The age-adjusted race disparity OR (i.e., the increase in odds of HTN for black compared to white women) was 3.3 (95% CI: 2.0–5.5) (Table 2). In the model adjusted for midlife SES (Model 2), the disparity OR was 2.5 (95% CI: 1.5–4.4)

(Table 2). Following adjustment for midlife hostility (Model 3), the disparity OR was 2.4 (95% CI: 1.4–4.3) (Table 2). After adjustment for midlife diet quality score and sedentary hours (Model 4), the disparity OR was 2.3 (95% CI: 1.3–4.1); and after adjustment for midlife BMI and CRP z-score (Model 5), the disparity OR was 2.1 (95% CI: 1.2–4.0) (Table 2). Adding pregnancy variables (pregnancy BMI, CRP, and depressive symptoms, Model 6) reduced the OR to 1.9, which was not significantly different from 0 (95% CI: 1.0–3.7) (Table 2). Replacing both midlife and prepregnancy BMI with change in BMI from prepregnancy to midlife did not substantially alter our findings (Table 2).

Findings for average blood pressure were similar to those for HTN and are presented in Table 3; in these models, the beta coefficient (SE, p -value) for black women represents the disparity in average blood pressure that would remain between black and white women if the distributions of other covariates in the black population were set equal to the distributions in the white population. As with HTN, adjustment for midlife SES strongly attenuated the age-adjusted race disparity from 6.2 (1.2, $p<0.01$) to 4.1 mmHg (1.1, $p<0.01$). Further adjustment for diet quality score and sedentary hours attenuated the disparity to 3.6 mmHg; and adjustment for BMI and CRP z-score attenuated the disparity to 2.7 mmHg (1.1, $p<0.05$). Inclusion of the pregnancy factors attenuated the race disparity to 2.2 mmHg (1.2, $p=0.06$).

There were no significant interactions between race and either midlife BMI ($p=0.40$) or age ($p=0.89$) in these models. Race was not strongly associated with pre-HTN; ORs comparing pre-HTN to normotension (from the multinomial model) are presented in Appendix Table A2. Findings from the complete case sample were substantively similar (e.g., age-adjusted OR [Model 1] was 3.4 [95% CI: 2.0–5.8]; full data available upon request).

Discussion

These data are the first, of which we are aware, to examine the contribution of risk factors measured both concurrently and at the time of pregnancy (i.e., 7 to 15 years earlier) to black-white disparities in HTN among women at midlife. We report three key findings. First, SES explained a substantial proportion of the disparity between black and white women in hypertensive status and average blood pressure. Second, although further adjustment for known psychosocial, behavioral, and physiological risk factors attenuated the race disparity in HTN and blood pressure, these established risk factors did not fully explain the race disparity. Third, inclusion of risk factors measured at pregnancy (i.e., prepregnancy BMI, depressive symptoms, and CRP) further attenuated the race disparity in both HTN and blood pressure to values not statistically significantly different from the null.

Our data—along with those from prior studies—indicate that factors beyond SES and health behaviors need to be considered to fully understand the drivers behind race disparities in HTN. By adding variables measured at pregnancy to our models, we sought to determine whether factors measured earlier in life—and at a pivotal point in a woman's health life course—could strengthen our understanding of racial disparities in HTN at midlife. Indeed, our data do suggest that health indicators measured at pregnancy, specifically prepregnancy BMI, depressive symptoms, and CRP

TABLE 1. DESCRIPTIVE ANALYSES OF FACTORS MEASURED AT FOLLOW-UP IN MIDLIFE AND AT THE TIME OF PREGNANCY IN POUCHMOMS COHORT, MICHIGAN, UNITED STATES (N=615)

	Overall	<i>n (%) or mean (SD)</i>		p-value ^a
		African American	White	
Race/ethnicity				
African American	242 (24.6)	—	—	
White/other	373 (75.4)	—	—	
Midlife variables				
Outcome variables				
Hypertensive status				<0.01
Normotensive	340 (57.3)	111 (45.1)	229 (61.3)	
Prehypertensive	149 (24.1)	63 (26.4)	86 (23.3)	
Hypertensive	126 (18.6)	68 (28.5)	58 (15.4)	
Continuous blood pressure				
Systolic	114.6 (14.1)	118.6 (15.9)	113.3 (12.2)	<0.01
Diastolic	75.5 (11.0)	78.7 (11.2)	74.5 (10.5)	<0.01
Average	95.1 (11.9)	98.6 (12.9)	93.9 (10.7)	<0.01
Risk factors				
Age	37.7 (5.6)	34.9 (4.9)	38.7 (5.6)	<0.01
Socioeconomic status				<0.01
High	170 (32.9)	22 (9.2)	158 (39.1)	
Moderate	239 (38.9)	82 (33.2)	174 (41.6)	
Low	206 (28.2)	138 (57.6)	78 (19.3)	
Depression ^b				0.08
No depression	399 (67.7)	163 (69.3)	236 (67.1)	
Major depression	173 (28.2)	65 (29.5)	108 (27.7)	
Short symptomatology	19 (4.2)	3 (1.2)	16 (5.2)	
Job strain ^b				0.20
Low strain	118 (26.8)	37 (24.8)	81 (27.3)	
Active	96 (21)	26 (15.6)	70 (22.4)	
Passive	143 (35.4)	55 (36.8)	88 (35)	
High strain	71 (16.9)	30 (22.8)	41 (15.3)	
Hostility ^b	4.2 (2.1)	5.2 (2.1)	3.9 (2.0)	<0.01
Current smoking				0.18
Yes	174 (27.8)	80 (31.9)	94 (26.4)	
No	441 (72.2)	162 (68.1)	279 (73.6)	
Diet quality score ^b	53.4 (10.8)	48.7 (10.0)	54.9 (10.5)	<0.01
Sedentary time (hrs/week)	46.5 (24.5)	52.2 (28.8)	44.6 (20.8)	<0.01
Sleep Score	8.8 (3.4)	8.5 (3.4)	8.9 (3.4)	0.18
Body mass index (kg/m ²)	31.2 (8.9)	33.6 (9.4)	30.5 (8.3)	<0.01
Lipids ^b				
HDL-C	54.5 (13.9)	53.7 (13.8)	54.8 (13.9)	0.38
LDL-C	106.1 (30.3)	107.9 (32.2)	105.5 (29.0)	0.39
Triglycerides	113.3 (87.3)	89.3 (41.6)	121.1 (104.8)	<0.01
C-reactive protein z-score ^b	0 (1.0)	0 (0.9)	0 (1.0)	0.71
Pregnancy variables				
Socioeconomic status ^c				<0.01
High	162 (32.2)	23 (10.7)	139 (38.6)	
Moderate	250 (43.6)	90 (40.9)	160 (44.4)	
Low	156 (24.2)	100 (48.4)	56 (17.0)	
Hostility ^c	4.2 (2.1)	5.4 (1.9)	3.8 (1.9)	<0.01
Depressive symptoms ^c	13.4 (9.7)	17.3 (10.7)	12.1 (8.5)	<0.01
Prepregnancy body mass index (kg/m ²)	27.1 (8.9)	28.0 (7.8)	26.9 (7.2)	0.12
Lipids ^c				
HDL-C	68.2 (15.3)	70.8 (15.4)	67.4 (15.2)	0.02
LDL-C	123.7 (38.5)	116.6 (35.6)	125.9 (39.7)	<0.01
Triglycerides	187.5 (72.9)	157.6 (41.6)	197 (75.3)	<0.01
C-reactive protein ⁴	7.3 (5.4)	7.6 (5.5)	7.2 (5.4)	0.41
Hypertensive disorders of pregnancy				<0.01
Normotensive	564 (92.1)	235 (97.1)	329 (90.5)	
Gestational hypertension or preeclampsia	51 (7.9)	7 (2.9)	44 (9.5)	

(continued)

TABLE 1. (CONTINUED)

	Overall	<i>n (%) or mean (SD)</i>		<i>p-value</i> ^a
		African American	White	
Gestational age at delivery				<0.01
Term (≥37 weeks)	465 (89.3)	195 (85.7)	270 (90.5)	
Preterm (<37 weeks)	150 (10.7)	47 (14.3)	103 (9.5)	

Analyses are weighted for subcohort sampling strata.

^a*p*-value comparing African American to white.

^bMissing values at midlife: depression (*n* = 25), job strain (*n* = 197), hostility (*n* = 27), diet quality (*n* = 1), HDL-C and triglycerides (*n* = 1), LDL-C and CRP z-score (*n* = 2).

^cMissing values at pregnancy: socioeconomic status (*n* = 50), hostility (*n* = 1), depressive symptoms (*n* = 2), lipids and CRP (*n* = 28).

HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

levels during pregnancy, were associated with HTN and blood pressure at midlife and contributed to the racial disparity in HTN at midlife. These three indicators of pregnancy health are notable in that they reflect multiple aspects of health—that is, body size, mental health, and inflammation.

Our findings concur with those of prior studies using data from the Coronary Artery Risk Development in Young Adults cohort and the National Health and Nutrition Examination Survey, but extend them in several important ways. Research using these national datasets reports that SES, behavioral risk factors, BMI, abdominal obesity, and other biomarkers contribute to, but do not fully account for, the black-white disparity.^{5,6,8,11} Our findings also show that SES is strongly associated with HTN and blood pressure at midlife, and adjustment for SES resulted in a large reduction in the race disparity measures. BMI was also an important

risk factor for HTN and contributed to the race disparity (particularly for average blood pressure) in our study. BMI likely acts as a mediator connecting more distal factors such as SES and psychosocial and behavioral factors to HTN.

On the other hand, although prior research has demonstrated strong associations between pregnancy complications and risk of HTN later in life, we did not find evidence that hypertensive disorders of pregnancy or PTD contributed to racial disparities in HTN in our data. In fact, black women in the POUCH Study had a lower prevalence of hypertensive disorders of pregnancy compared to white women (perhaps due to black mothers being on average younger at pregnancy), and although PTD was more prevalent among black (vs. white) women, it was not strongly associated with HTN in our data. Thus, while we did not find that the established pregnancy complications were related to the race disparity in

TABLE 2. ASSOCIATIONS BETWEEN RACE/ETHNICITY AND HYPERTENSIVE (COMPARED TO NORMOTENSIVE) WITH PROGRESSIVE ADJUSTMENT FOR MIDLIFE AND PREGNANCY HEALTH FACTORS IN POUCHMOM STUDY (*N* = 615)

	<i>OR (95% CI)</i>					
	<i>Age-adjusted model 1</i>	<i>Midlife SES model 2</i>	<i>Midlife psychosocial model 3</i>	<i>Midlife behavioral model 4</i>	<i>Midlife physiological model 5</i>	<i>Pregnancy model 6</i>
Race/ethnicity						
African American	3.3 (2.0–5.5)	2.5 (1.5–4.4)	2.4 (1.4–4.3)	2.3 (1.3–4.1)	2.1 (1.2–4.0)	1.9 (1.0–3.7)
White/other	ref	ref	ref	ref	ref	ref
Age	1.1 (1.0–1.1)	1.1 (1.0–1.2)	1.1 (1.0–1.2)	1.1 (1.0–1.2)	1.1 (1.0–1.2)	1.1 (1.0–1.2)
Midlife risk factors						
Socioeconomic status						
High	—	ref	ref	ref	ref	ref
Moderate	—	2.6 (1.3–5.2)	2.7 (1.3–5.6)	2.7 (1.3–5.6)	2.0 (0.9–4.3)	2.1 (0.9–4.8)
Low	—	3.1 (1.5–6.8)	3.0 (1.3–6.8)	2.9 (1.3–6.5)	2.2 (0.9–5.3)	2.4 (0.9–6.5)
Hostility	—	—	1.1 (1.0–1.2)	1.1 (0.9–1.2)	1.0 (0.9–1.2)	1.0 (0.9–1.2)
Diet quality score	—	—	—	1.0 (1.0–1.0)	1.0 (1.0–1.0)	1.0 (1.0–1.0)
Sedentary time (hrs/week)	—	—	—	1.0 (1.0–1.0)	1.0 (1.0–1.0)	1.0 (1.0–1.0)
Body mass index (kg/m ²)	—	—	—	—	1.1 (1.1–1.2)	1.2 (1.1–1.2)
C-reactive protein z-score	—	—	—	—	0.9 (0.6–1.2)	0.7 (0.5–1.1)
Pregnancy risk factors						
Prepregnancy body mass index (kg/m ²)	—	—	—	—	—	1.0 (0.9–1.0)
C-reactive protein	—	—	—	—	—	1.7 (1.1–2.7)
Depressive symptoms	—	—	—	—	—	1.0 (1.0–1.1)

Analyses are weighted for subcohort sampling strata and reflect odds ratios from multinomial model with 3-part outcome (hypertensive, prehypertensive [results in Appendix Table A1], and normotensive [reference]).

“—” indicates that the variable was not included in the model.

TABLE 3. ASSOCIATIONS BETWEEN RACE/ETHNICITY AND AVERAGE BLOOD PRESSURE (MMHG) WITH PROGRESSIVE ADJUSTMENT FOR MIDLIFE AND PREGNANCY HEALTH FACTORS IN POUCHMOM STUDY (N=615)

	β (SE)					
	Age-adjusted model 1	Midlife SES model 2	Midlife psychosocial model 3	Midlife behavioral model 4	Midlife physiological model 5	Pregnancy model 6
Race/ethnicity						
African American	6.2 (1.2) ^a	4.1 (1.1) ^a	4.2 (1.2) ^a	3.6 (1.2) ^a	2.7 (1.1) ^b	2.2 (1.2)
White/other	ref	ref	Ref	ref	ref	ref
Age	0.4 (0.1) ^a	0.6 (0.1) ^a	0.6 (0.1) ^a	0.6 (0.1) ^a	0.6 (0.1) ^a	0.6 (0.1) ^a
Midlife risk factors						
Socioeconomic status						
High	—	ref	Ref	ref	ref	ref
Moderate	—	7.2 (1.3) ^a	7.2 (1.4) ^a	6.6 (1.4) ^a	4.9 (1.3) ^a	4.7 (1.3) ^a
Low	—	8.2 (1.5) ^a	8.0 (1.6) ^a	7.2 (1.6) ^a	5.9 (1.5) ^a	6.1 (1.6)
Hostility	—	—	0.2 (0.3)	0.1	−0.1 (0.2)	−0.2 (0.3)
Diet quality score	—	—	—	−0.1 (0.1) ^b	−0.1 (0.1)	−0.1 (0.1)
Sedentary time (hrs/week)	—	—	—	0.1 (0.0) ^b	0.0 (0.0)	0.0 (0.0)
Body mass index (kg/m ²)	—	—	—	—	0.5 (0.1) ^a	0.6 (0.6) ^a
C-reactive protein z-score	—	—	—	—	−0.4 (0.5)	−0.8 (0.5)
Pregnancy risk factors						
Prepregnancy body mass index (kg/m ²)	—	—	—	—	—	−0.2 (0.1)
C-reactive protein	—	—	—	—	—	1.3 (0.5) ^b
Depressive symptoms	—	—	—	—	—	0.1 (0.1)

Analyses are weighted for subcohort sampling strata.

“—” indicates that the variable was not included in the model.

^a $p < 0.01$.

^b $p < 0.05$.

HTN in our data, we did find evidence that general indicators of health during pregnancy were related to race disparities in HTN at midlife.

We acknowledge that our study did not include data on other potentially important factors that might contribute to the race disparity in HTN, such as genetics, racial discrimination, or neighborhood environment. However, we aimed to focus on the most well-established and commonly assessed individual-level risk factors for HTN and to then add factors measured at pregnancy to these variables. It also remains possible that we did not measure all important psychosocial, behavioral, or physiological factors; however, we believe that we have included the majority of factors in each category shown to be associated with CVD in prior work. We were also unable to include measures of change in participant income over time, life stress, or racial discrimination, although these may be salient factors as indicated in Figure 1. We also note that variables such as prepregnancy BMI and socioeconomic position were based on self-report. Moreover, our findings cannot indicate whether the specific factors of prepregnancy BMI, depressive symptoms, and CRP are causal determinants of racial disparities in HTN. Finally, we acknowledge that the order in which variables are entered into sequential models can alter the apparent importance of those variables (e.g., BMI may have appeared more strongly associated with HTN if it were entered into the model first.) Thus, we relied on our conceptual framework outlined in Figure 1 to determine *a priori* the order of sequential models (i.e., distal to proximate) instead of basing these decisions on the data alone. We also acknowledge that our conceptual framework does not reflect the timing of risk factors (i.e.,

pregnancy or midlife), but we chose to present the simpler figure in favor of interpretability.

A notable aspect of our study is the fact that women were assessed in midlife, before the onset of most cardiovascular events; thus, we had an opportunity to identify race disparities in cardiovascular risk before the onset of clinical CVD. Further strengths include the use of standardized measures of blood pressure and risk factors (e.g., the CIDI, the Block Food Frequency Questionnaire). Finally, the POUCHMoms study is unique in that it includes extensive psychosocial and biomarker data from pregnancy as well as at the midlife follow-up in a cohort with a large proportion of both black and white women.

Our study makes an important contribution to our understanding of race disparities in HTN. Our results indicate that pregnancy health may portend future disparities in cardiovascular health. Our findings provide further evidence for the concept that pregnancy may serve as a “window” into women’s cardiovascular health later in life and into differences in cardiovascular health by race. In future studies, we hope to develop more refined risk-stratification tools that can identify pregnant women who would benefit from closer monitoring and targeted interventions postpregnancy to prevent progression to HTN. This strategy may be one of many employed to reduce race disparities in CVD among women.

Acknowledgments

The authors thank the POUCH and POUCHMoms community research study nurses for careful data collection and

Bertha Bullen and Jia Xu for their role in study logistics and data management.

The POUCH Study was supported by the Perinatal Epidemiological Research Initiative Program Grant from the March of Dimes Foundation (20FY01-38 and 20FY04-37), the Eunice Kennedy Shriver National Institute for Child Health and Human Development and the National Institute of Nursing Research (R01-HD34543), the Thrasher Research Foundation (02816-7), and the Centers for Disease Control and Prevention (U01-DP000143-01). The POUCHmoms Study was supported by the National Heart, Lung, and Blood Institute (NHLBI) (R01-HL103825). Dr. Margerison is supported by NHLBI (K01-HL128843).

Author Disclosure Statement

No competing financial interests exist.

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Appendix

APPENDIX TABLE A1. JOB STRAIN QUESTIONS BASED ON THE KARASEK DEMAND-CONTROL QUESTIONNAIRE^{A1}

Questions related to decision latitude

- a. My job requires that I learn new things.
- b. My job involves a lot of repetitive work. (reverse coded)
- c. My job requires me to be creative.
- d. My job allows me to make a lot of decisions on my own.
- e. My job requires a high level of skill.
- f. On my job, I have little freedom to decide how to do my work. (reverse coded)
- g. I get to do a variety of different things on my job.
- h. I have a lot of say about what happens on my job.
- i. I have an opportunity to develop my own special skills.

Questions related to demand

- j. My job requires working very fast
 - k. My job requires working very hard
 - l. I am not asked to do an excessive amount of work. (reverse coded)
 - m. I have enough time to get the job done. (reverse coded)
 - n. I am free from conflicting demands that others make. (reverse coded)
 - o. My supervisor is concerned about the welfare of those under him/her.
 - p. My supervisor pays attention to what I am saying.
 - q. People I work with are competent in doing their jobs.
 - r. People I work with take a personal interest in me.
 - s. People I work with are friendly.
 - t. People I work with are helpful in getting the job done.
-

All questions are answered on a scale of 1 to 4 (1=strongly disagree, 2=disagree, 3=agree, and 4=strongly agree).

APPENDIX TABLE A2. ASSOCIATIONS BETWEEN RACE/ETHNICITY AND PREHYPERTENSIVE (COMPARED TO NORMOTENSIVE) WITH PROGRESSIVE ADJUSTMENT FOR CONCURRENT RISK FACTORS IN POUCHMOM COHORT (N=615)

	OR (95% CI)					
	<i>Age-adjusted model 1</i>	<i>Midlife SES model 2</i>	<i>Midlife psychosocial model 3</i>	<i>Midlife behavioral model 4</i>	<i>Midlife physiological model 5</i>	<i>Pregnancy model 6</i>
Race/ethnicity						
African American	1.6 (1.0–2.6)	1.5 (0.9–2.4)	1.4 (0.8–2.3)	1.3 (0.8–2.2)	1.2 (0.7–2.1)	1.1 (0.6–2.0)
White/other	ref	ref	ref	ref	ref	ref
Age	1.0 (1.0–1.1)	1.0 (1.0–1.1)	1.0 (1.0–1.1)	1.0 (1.0–1.1)	1.0 (1.0–1.1)	1.0 (1.0–1.1)
Midlife risk factors						
Socioeconomic status						
High	—	ref	ref	ref	ref	ref
Moderate	—	2.0 (1.1–3.7)	2.0 (1.1–3.7)	1.9 (1.0–3.6)	1.5 (0.8–2.9)	1.5 (0.8–2.8)
Low	—	1.6 (0.8–3.1)	1.9 (0.9–3.8)	1.7 (0.8–3.6)	1.5 (0.7–3.1)	1.5 (0.7–3.4)
Hostility	—	—	1.0 (0.8–1.1)	0.9 (0.8–1.1)	0.9 (0.8–1.1)	0.9 (0.8–1.1)
Diet quality score	—	—	—	1.0 (1.0–1.0)	1.0 (1.0–1.0)	1.0 (1.0–1.0)
Sedentary time (hrs/week)	—	—	—	1.0 (1.0–1.0)	1.0 (1.0–1.0)	1.0 (1.0–1.0)
Body mass index (kg/m ²)	—	—	—	—	1.1 (1.0–1.1)	1.1 (1.1–1.2)
C-reactive protein z-score	—	—	—	—	1.0 (0.8–1.4)	1.0 (0.7–1.3)
Pregnancy risk factors						
Prepregnancy body mass index (kg/m ²)	—	—	—	—	—	0.9 (0.9–1.0)
C-reactive protein	—	—	—	—	—	1.3 (0.9–1.9)
Depressive symptoms	—	—	—	—	—	1.0 (1.0–1.1)

Analyses are weighted for subcohort sampling strata and reflect odds ratios from multinomial model with 3-part outcome (hypertensive, prehypertensive [results in Appendix Table A1], and normotensive [reference]).

“—” indicates that the variable was not included in the model.

Appendix Reference

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