

Original Article

Neighborhood Deprivation, Perceived Stress, and Pregnancy-Related Hypertension Phenotypes a Decade Following Pregnancy

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BACKGROUND: Hypertensive disorders in pregnancy and other adverse pregnancy outcomes (APOs) increase the risk of developing chronic hypertension and cardiovascular disease. Perceptions of stress and neighborhood context also influence blood pressure (BP) fluctuations. We examined if APOs, higher perceived stress, and neighborhood deprivation were associated with hypertension phenotypes a decade after pregnancy in untreated individuals.

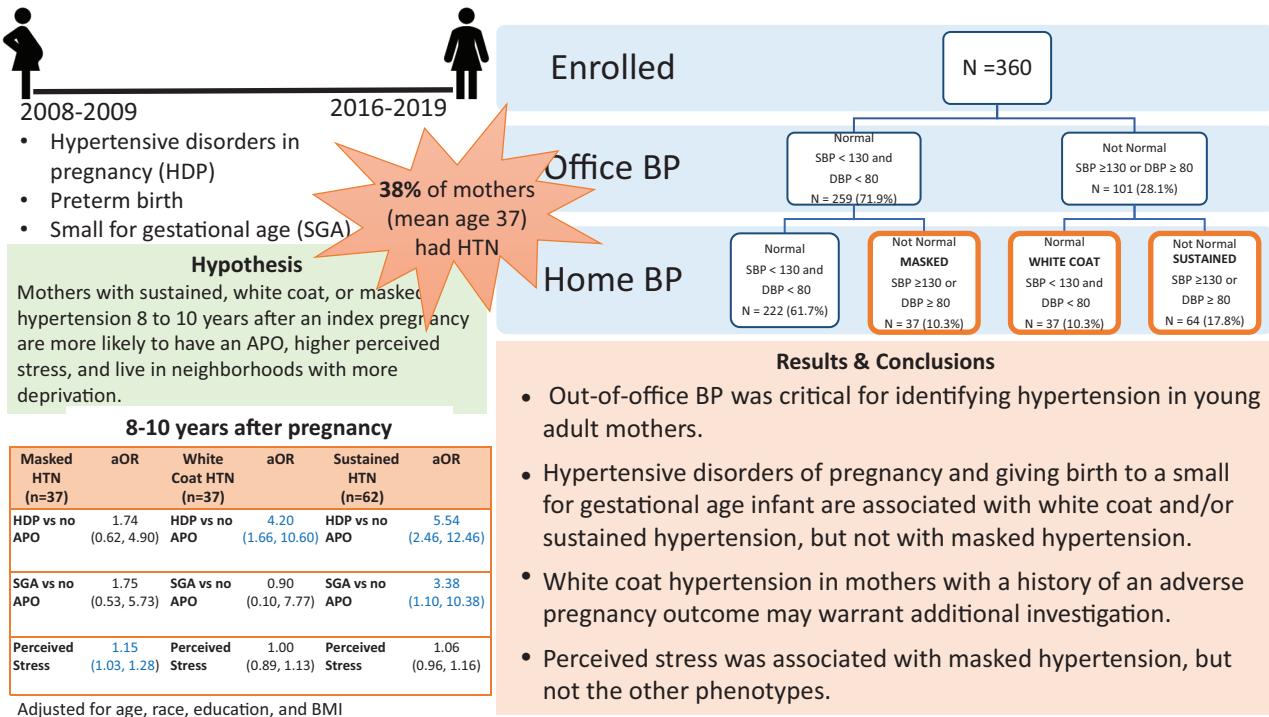
METHODS: Participants were 360 individuals who gave birth between 2008 and 2009 and participated in a research study 8–10 years following pregnancy. Standardized office and home BP readings were obtained, and we applied the AHA/ACC 2017 guidelines to identify sustained, white coat, and masked hypertension phenotypes. We measured personal stress with the perceived stress scale and neighborhood deprivation with the CDC Social Vulnerability Index.

RESULTS: Of the 38.3% (138/360) with any hypertension, 26.1% (36/138) reported a diagnosis of hypertension but were currently untreated. Sustained hypertension was the most common (17.8%), followed by masked and white coat hypertension, both 10.3%. Hypertensive disorders in pregnancy were associated with sustained (odds ratio [OR] 5.54 [95% confidence interval, CI 2.46, 12.46]) and white coat phenotypes (OR 4.20 [1.66, 10.60]), but not masked hypertension (OR 1.74 [0.62, 4.90]). Giving birth to a small for gestational age infant was also associated with sustained hypertension. In covariate adjusted models, perceived stress, but not neighborhood deprivation, was significantly associated with masked hypertension.

CONCLUSIONS: A decade after delivery, APOs were associated with sustained and white coat hypertension, but not masked hypertension. Exploration of the mechanisms underlying, and clinical implications of, these associations is warranted.

Keywords: blood pressure; chronic stress; hypertension; neighborhood factors; pregnancy; women's health.

Graphical Abstract



Hypertensive disorders of pregnancy (HDP), delivering a small for gestational age (SGA) infant, and preterm birth (PTB) are associated with an increased risk of future maternal cardiovascular disease (CVD).¹⁻⁶ Giving birth to an SGA infant can be related to placental insufficiency and many PTBs are attributed to vascular dysfunction and inflammation, a commonality between adverse pregnancy outcomes (APOs), often referred to as “great obstetrical syndromes.”¹ Because of the mechanistic pathways underlying many APO, pregnancy is considered a preview of the future cardiovascular health of the mother.

The cardiovascular risk associated with APO persists for decades, and chronic hypertension is a common first step in the progression to CVD.¹⁻⁶ In a cohort of first-time mothers, preeclampsia and PTB were associated with a nearly 3-fold higher risk of developing hypertension within 7 years after birth than women without pregnancy complications.³ Although the association between APOs and hypertension persists for decades,² the research evaluating out-of-office hypertension profiles after an APO has focused exclusively on HDP and the first year after birth.^{7,8} Underdiagnosed hypertension which occurs with masked hypertension could be a missed opportunity to manage one of the most significant risk factors for future CVD in young mothers. Moreover, hypertension profiles identified with home blood pressure monitoring (HBPM)-sustained, white coat, and masked hypertension are all associated with increased risk of cardiovascular events and mortality.⁹⁻¹¹

Identifying hypertension phenotypes and their relationship with pregnancy and other modifiable risk factors is important for risk stratification and future interventions. Environmental factors such as neighborhood-level poverty, decreased walkability, and availability of healthy food increase risk of hypertension and CVD mortality.¹²⁻¹⁶ Individuals residing in neighborhoods with more deprivation often report more stress, an individual factor also associated with hypertension.¹² Interestingly, the association

of neighborhood factors with white coat or masked hypertension phenotypes has not been evaluated, an essential next step to identify targets for multilevel interventions (individual, community, and policy) and to respond to calls for targeted interventions for mothers with a history of APOs.¹⁷

The purpose of this study was (i) to describe the prevalence of hypertension phenotypes (sustained, white coat, and masked hypertension) in women 10 years after an index pregnancy and (ii) to test the hypothesis that compared with women with normal blood pressure (BP), women with a hypertension phenotype 8–10 years after an index pregnancy are more likely to have a history of an APO, higher perceived stress, and live in neighborhoods with a higher deprivation index.

METHODS

The current study is a secondary analysis of data from a clinical cohort of 505 women who gave birth between 2008 and 2009 in southwest Pennsylvania, identified as the index pregnancy and then recontacted 8–10 years later. Eligibility for the parent study included age 18 years or above, placental pathology evaluated at time of delivery, and not currently pregnant. Microscopic placental evaluation is indicated for certain neonatal or maternal complications such as PTB, HDP, fetal growth restriction, meconium-stained amniotic fluid, and placental type (e.g., placenta previa).^{18,19} Exclusion criteria included preexisting diagnosis of chronic hypertension determined by a medical record review. Potential participants meeting the inclusion and exclusion criteria were identified and contacted between 2016 and 2019. Of the 505 women who consented to participate in the parent study, the current analysis includes 360 women who were not taking an antihypertensive and had home BP assessments and census tract data available (Figure 1). The secondary analysis of deidentified data was determined to be exempt by the University of South

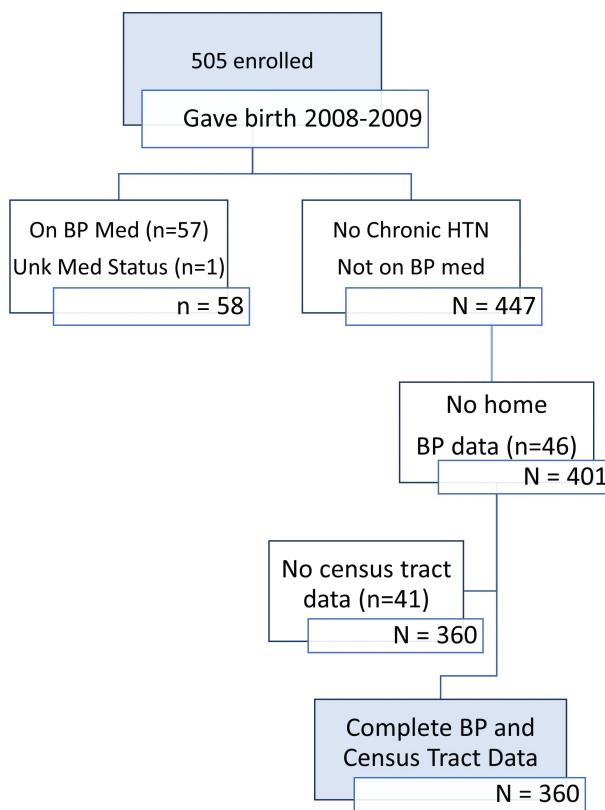


Figure 1. Flowchart of data available for analysis.

Carolina IRB. The data underlying this article may be shared on reasonable request to the corresponding author.

Outcome variable

We determined hypertension phenotypes based upon office and home BP assessed at the research visit conducted in 2016–2019. BP was measured 3 times using a validated device (Microlife A6 PC/BP3GUI-8X) and following the research protocol including feet rested on the floor, seated for 5 minutes before BP measurement, and measurement of arm circumference to identify the appropriate cuff size. The mean systolic and diastolic blood pressure were calculated. Participants were then trained in the use of the home BP monitor (same monitor as the office visit) by a trained research staff member and instructed to check their BP twice a day over the course of a week.²⁰ Most participants (96.7%, 348/360) recorded 4 or more BPs over the 7-day period.

Hypertension was defined as systolic blood pressure ≥ 130 or diastolic blood pressure ≥ 80 consistent with the current national guidelines.²¹ We classified masked, white coat, and sustained hypertension phenotypes according to the mean office BP and mean home BP measurements (Figure 2).²¹

Exposure variables

APOs—HDP, PTB, and SGA—were assessed from the electronic medical record. HDP were identified based on new BP elevations after 20 weeks gestation and includes individuals with evidence of end-organ involvement, such as proteinuria, and those without.²² PTB was defined as a birth <37 weeks gestation and included both spontaneous and iatrogenic PTB, and SGA infant was determined using a Global Reference for weight percentiles that accounts for gestational age.²³ For the analysis, the 3 APO categories are mutually exclusive, as shown in Figure 3.

Neighborhood deprivation was measured 8–10 years post birth using the 2018 CDC Social Vulnerability Index consisting of 4 domains (i.e., socioeconomic status, household composition, minority status, and housing type) measured at the census tract level (Supplementary Figure S1 online). Each census tract is assigned a rank (0–1) relative to the other census tracts in Pennsylvania, with higher values representing greater deprivation. The index is included in the NIH Structural Determinants of Health PhenX toolkit and has been used to link neighborhood deprivation with health factors.^{12,13,24,25}

The perceived stress scale assesses individual-level perceptions of stress in the past month and a higher value represents greater stress levels.²⁶ The perceived stress scale is included in the NIH PhenX toolkit as a validated psychosocial measure and demonstrated good reliability in our sample ($\alpha = 0.76$).²⁵

Covariates were self-reported age, racial identity, highest education attained, and objectively measured body mass index. Body mass index was calculated from the weight and height measurements at the research visit.

Analysis

The characteristics of the sample were summarized using descriptive statistics and scatterplots. Data are expressed as mean \pm SD. The distribution of data was assessed using histograms and Kolmogorov-Smirnov test for normality. Differences in APO, neighborhood, and individual factors by hypertension phenotype were tested using Chi-square with Fisher correction as indicated or 1-way ANOVA for continuous variables. Bonferroni-adjusted post hoc tests were used to examine the pairwise differences between phenotype groups ($0.05/6 = 0.008$). If the assumptions of ANOVA were not met, a nonparametric Kruskal-Wallis with Wilcoxon Rank Sum post hoc test were used.

To test the hypothesis that APO, higher neighborhood deprivation, or perceived stress would be more likely to have an abnormal hypertension phenotype, a separate multivariable logistic regression analyses was conducted for each hypertension phenotype relative to the normotensive group. An α -level of 0.05 was selected to indicate statistical significance. Education was categorized as college graduate (yes vs. no) and racial identity was categorized as Black or African American (yes vs. no). The first model included the APO, neighborhood deprivation, and perceived stress. Older age, racial stress (self-identified race as a proxy), lower education, and higher body mass index are accounted for in the second model.^{27,28} All analyses were conducted in SAS v9.4 and all available data were used without imputation.

RESULTS

At 9.2 ± 0.9 years after the index pregnancy, the women ranged in age from 25 to 51 years (mean 37.7 ± 5.9), 48.3% were college graduates, most had overweight or obesity (69%), and 43.3% had an APO (Table 1). HDP was the most common APO (69/360). There were 11 cases of preeclampsia in another pregnancy, but these were too few to analyze separately. The overall neighborhood deprivation score ranged from 0.0003 to 0.997 (higher values represent greater deprivation), with a mean of 0.457 (± 0.31).

Overall, 38.3% of the women had an abnormal hypertension phenotype 8–10 years after pregnancy (Figure 2). The normotensive phenotype was most common ($N = 222$, 61.7%), followed by

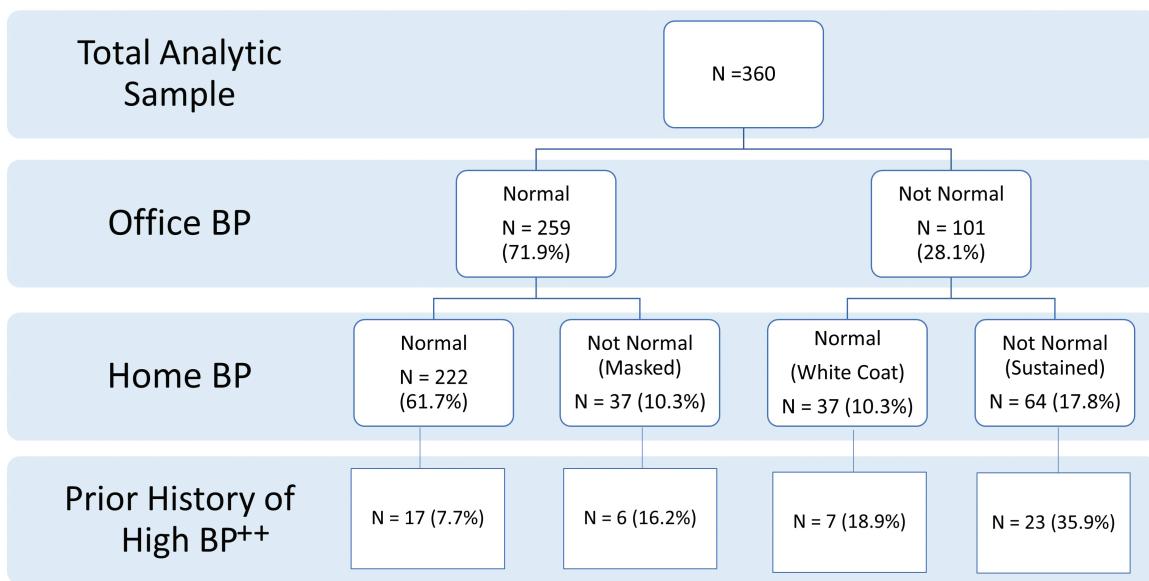


Figure 2. Identification of hypertension phenotypes. Abbreviation: BP, blood pressure. ++% reported is % of the total in the preceding row.

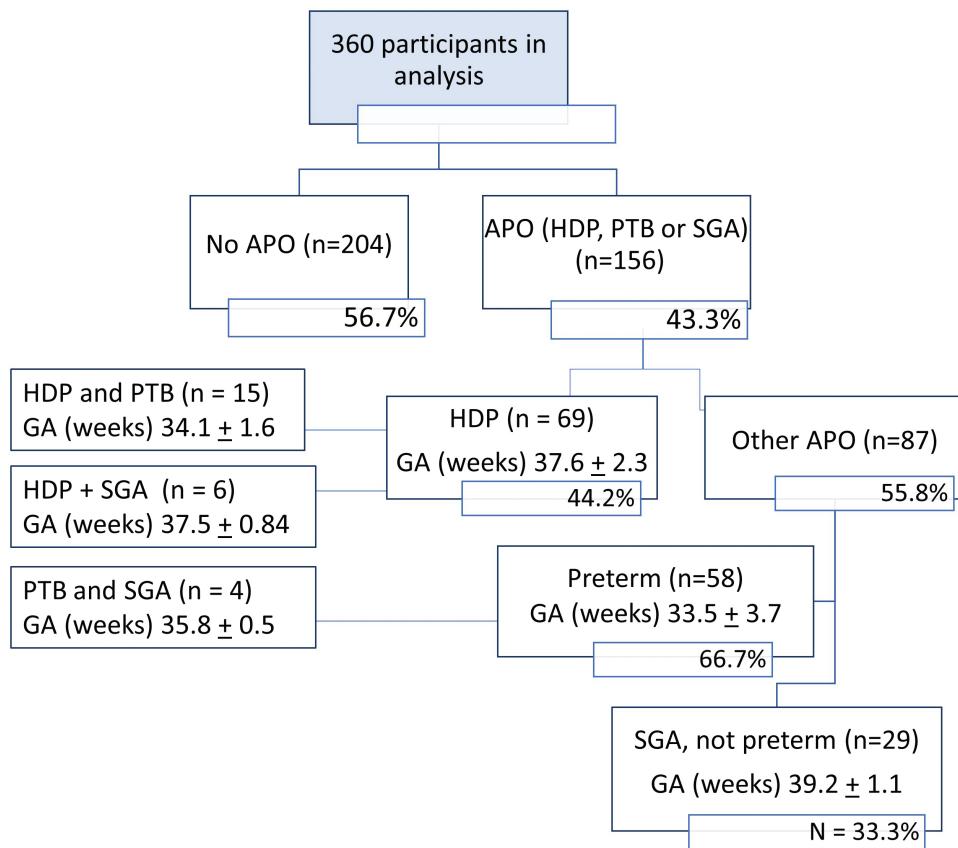


Figure 3. Prevalence of adverse pregnancy outcomes in the study sample. Abbreviations: APO, adverse pregnancy outcome; GA, gestational age; HDP, hypertensive disorder in pregnancy; PTB, preterm birth; SGA, small for gestational age.

sustained hypertension (N = 64, 17.8%) and masked and white coat hypertension (both with N = 37, 10.3%). A quarter of the women with an abnormal hypertension phenotype reported a prior history of hypertension but were not taking an antihypertensive (N = 36/138).

APO and neighborhood deprivation by hypertension phenotype

Compared with the normotensive phenotype, the women in the masked hypertension phenotype were younger (34.8 ± 6.2 vs. 37.9 ± 5.7 , $P = 0.017$) and a greater proportion identified as

Table 1. Sample characteristics of total sample and by hypertension phenotype (N = 360)

Pregnancy factors	N	Total sample N = 360	n (%) or m ± SD	Normotensive n = 222	Masked HTN n = 37	White coat HTN n = 37	Sustained HTN n = 64	n (%) or m ± SD	n (%) or m ± SD	P
Hypertensive disorder of pregnancy ^b	360	69 (19.17)	25 (11.26)	8 (21.62)	14 (37.84)	22 (34.38)	<0.0001			
HD/P + PTB ^{a,b}		15 (4.17)	5 (2.25)	2 (5.41)	2 (5.41)	6 (9.38)	0.007			
HD/P + SGA, not PTB ^{a,b}		6 (1.67)	3 (1.35)	1 (2.70)	0	2 (3.13)	0.264			
PTB (n, %) ^b	360	58 (16.11)	38 (17.12)	5 (13.51)	5 (13.51)	10 (15.63)	0.798			
PTB + SGA ^{a,b}		4 (1.11)	3 (1.35)	0	0	1 (1.56)	0.754			
SGA (n, %) ^{a,b}	360	29 (8.06)	15 (6.76) ³	6 (16.22) ³	1 (2.70)	7 (10.94)	0.042			
Any adverse pregnancy outcome (n, %) ^b	360	156 (43.33)	78 (35.14)³	19 (51.35)	20 (54.05) ³	39 (60.94)	0.001			
Follow-up 8–10 years post pregnancy	360	115.43 ± 13.72	109.02 ± 8.64	113.16 ± 7.55	125.79 ± 8.81	133.01 ± 14.45				
Office—systolic blood pressure	360	74.81 ± 9.87	69.63 ± 5.67	72.89 ± 4.98	82.99 ± 3.68	89.18 ± 8.63	<0.0001			
Office—diastolic blood pressure	360	117.11 ± 11.63	111.69 ± 8.17	126.78 ± 8.41	114.59 ± 7.37	131.78 ± 9.20	<0.0001			
Home—systolic blood pressure	360	75.32 ± 8.61	70.72 ± 5.40	84.49 ± 4.58	73.91 ± 3.64	86.82 ± 6.41	<0.0001			
Individual sociodemographic factors										
Age in years	353	37.72 ± 5.86	37.91 ± 5.71 ³	34.81 ± 6.22 ³	38.89 ± 5.80 ³	38.10 ± 5.79	0.011			
Education	360	74 (20.56)	39 (17.57)	15 (40.54)	5 (13.51)	15 (23.44)	0.000			
≤ High school		112 (31.11)	58 (26.13)	14 (37.84)	14 (37.84)	26 (40.63)				
Associate's or Trade School		174 (48.33)	125 (56.31)	8 (21.62)³	18 (48.65) ³	23 (35.94)				
College graduate										
Self-reported race										
Black	360	113 (31.39)	55 (24.77)	22 (59.46)¹	7 (18.92) ^{1,2}	29 (45.31)²	<0.0001			
White		242 (67.22)	163 (73.42)	15 (40.54)	30 (81.08)	34 (53.13)				
Racial identity is not Black or White ^c	351	5 (1.39)	4 (1.80)	0	0	1 (1.56)				
Visit BMI		29.76 ± 7.69	28.01 ± 7.13	29.60 ± 5.74	34.09 ± 9.80	33.30 ± 7.06	<0.0001			
Neighborhood deprivation										
Overall ranking (1 = greatest deprivation)	359	0.457 ± 0.31	0.423 ± 0.31	0.613 ± 0.29³	0.396 ± 0.29 ³	0.519 ± 0.31	0.002			
Socioeconomic status	359	0.465 ± 0.30	0.431 ± 0.30	0.629 ± 0.26³	0.415 ± 0.30 ³	0.519 ± 0.29	0.001			
Household composition and disability	359	0.466 ± 0.33	0.451 ± 0.33	0.553 ± 0.32	0.448 ± 0.31	0.481 ± 0.36	0.355			
Minority status and language	359	0.509 ± 0.23	0.498 ± 0.23	0.571 ± 0.21	0.470 ± 0.23	0.534 ± 0.25	0.212			
Housing type and transportation	359	0.455 ± 0.29	0.429 ± 0.28 ³	0.560 ± 0.28 ³	0.380 ± 0.28 ³	0.531 ± 0.31	0.004			
Perceived stress (m, SD)	360	5.02 ± 3.39	4.60 ± 3.07 ³	6.62 ± 3.95 ³	5.05 ± 3.45	5.50 ± 3.79	0.024			
Follow-up in years (m, SD)	360	9.15 ± 0.90	9.08 ± 0.90	9.38 ± 0.74	9.08 ± 0.89	9.28 ± 0.95	0.142			
Self-reported prior history of high BP	360	53 (14.72)	17 (7.66)	6 (16.22) ³	7 (18.92)	23 (35.94)³	<0.0001			

Abbreviations: BMI, body mass index; BP, blood pressure; HD/P, hypertensive disorder of pregnancy; HTN, hypertension; PTB, preterm birth; SGA, small for gestational age infant. Bolded = the phenotype is significantly different from the normotensive group and P ≤ 0.008, 1,2 = the 2 phenotypes are significantly different from each other and P < 0.05.

^aFisher's exact Chi-square

^bComparison group = no adverse pregnancy outcome.

^cRacial identity is not Black or White was analyzed with the group identifying as White, Bonferroni-adjusted P value = 0.05/6 = 0.008.

Black (59.5% vs. 24.8%, $P < 0.001$), and were not college graduates (21.6% vs. 56.3%, $P < 0.001$). Women in the white coat and sustained hypertensive phenotypes had significantly higher body mass index than the normotensive phenotype (34.1 ± 9.8 , 33.3 ± 7.1 vs. 28.0 ± 7.1 , $P = 0.001$).

A greater proportion of women with HDP were in the sustained or white coat hypertension phenotype than the normotensive phenotype (34.4%, 37.8% vs. 11.3%, $P < 0.001$, respectively). For PTB, there were no significant differences in pregnancy factors ($P = 0.798$). However, there was a higher proportion of those with iatrogenic PTB with sustained hypertension than were normotensive (9.38% vs. 2.25%, $P = 0.004$; see [Supplementary Table S1](#) online). Notably, women who birthed an SGA infant that was not due to a PTB or HDP ([Supplementary Figure S1](#) online) tended to be in the masked (16.2% vs. 6.8%, $P = 0.038$) or sustained hypertension (10.9% vs. 6.8%, $P = 0.064$) groups compared with the normotensive group.

Compared with the normotensive phenotype, women in the masked hypertension group resided in neighborhoods with greater overall deprivation (0.61 ± 0.29 vs. 0.42 ± 0.31 , $P = 0.005$) and lower socioeconomic status (0.63 ± 0.26 vs. 0.43 ± 0.30 , $P = 0.001$). No other significant differences in neighborhood deprivation were observed.

Predictors of hypertension phenotypes

HDP and birthing an SGA infant were associated with greater odds of sustained hypertension relative to normotensive (HDP: odds ratio [OR] 5.4 [95% confidence interval, CI 2.46, 12.46], SGA: OR 3.38 [95% CI 1.10, 10.38]) ([Table 2](#)). Neighborhood deprivation was not associated with greater odds of sustained hypertension. A sensitivity analysis was conducted to determine if the relationship of SGA with sustained hypertension may be explained by preeclampsia in a pregnancy before or after the index pregnancy. Adjusting for a history of preeclampsia in other pregnancies did not alter the findings ([Supplementary Table S2](#) online).

Women with the masked hypertension phenotype were more likely to reside in neighborhoods with more deprivation (OR 4.28 [95% CI 1.24, 14.74]) and reported higher perceived stress (OR 1.16 [95% CI 1.04, 1.29] per unit increase in reported stress) relative to the normotensive phenotype, however only perceived stress remained significant after accounting for individual socio-demographic factors (OR 1.15 [95% CI 1.03, 1.28]). None of the APO were significantly associated with the masked hypertension phenotype. Adjusting for self-reported prior history of hypertension or excluding participants with fewer than 4 home measurements did not significantly alter the interpretation of findings ([Supplementary Table S3](#) online).

To better understand the masked hypertension group, we reviewed scatterplots of the mean office and home BP by phenotype ([Supplementary Figure S2](#) online). For the masked hypertension phenotype, there were fewer home systolic elevations (red triangles) than home diastolic elevations (yellow "x"). Of the 37 women with masked hypertension, 70.3% ($N = 26$) had masked—isolated diastolic hypertension. In the sustained hypertension group, nearly all had systolic and diastolic hypertension ($N = 60/64$, 93.8%).

DISCUSSION

We conducted one of the few studies to examine how pregnancy-related factors may have a persistent influence on office and home-based BPs, which we categorized into phenotypes. A decade after pregnancy, we found that nearly 40% did not

have a normal BP phenotype. It is likely that the diagnosis of hypertension due to masked hypertension would be undetected in 11% of the sample of young women without HBPM. The masked hypertension phenotype was the only phenotype not associated with APO; however, it was associated with living in a neighborhood with greater deprivation and higher perceived stress.

Our findings that APO have a strong relationship with the sustained hypertension phenotype are consistent with prior evidence that some APO are an early indicator of future CVD risk.^{1,2,4,6} The association of SGA and sustained hypertension is consistent with other research identifying SGA as an indicator of underlying vascular dysfunction, a harbinger of future CVD risk for the mother and the offspring.¹ Of note, our findings supported SGA as a possible independent contributor to sustained hypertension after accounting for the risk associated with HDP in any past pregnancy.

We also found a robust association between HDP and the white coat hypertension phenotype, which is not initially intuitive. A substantial body of literature shows that white coat hypertension is not a benign phenotype due to associations with arterial stiffness, end-organ damage (e.g., microalbuminuria), cardiovascular events (e.g., nonfatal MI), and worse perinatal outcomes, similar to sustained hypertension.^{9–11,29} Due to their elevated BP in the office setting, they are more likely to receive treatment than the masked hypertension group that presents in the office with normal BP.¹¹ Of note, most research on white coat hypertension phenotype does not include data on pregnancy history.²⁹ As a result, the significance of the white coat phenotype after an APO remains unclear. We hypothesize that this group likely has a similarly elevated CVD risk as sustained hypertension, but this is a testable hypothesis for future work.

Preeclampsia is associated with masked hypertension within 1 year of pregnancy, but we did not find an association 8–10 years after pregnancy.^{7,8} It is plausible that a proportion of women with APO and masked hypertension would have transitioned to sustained (i.e., chronic) hypertension in the interval between the first postpartum year and 8–10 years postpartum.³ This may be especially true among Black women because of this population's earlier onset of hypertension.^{30,31} Additionally, of the 3 phenotypes, masked hypertension had the smallest proportion to report a prior diagnosis of hypertension. This is consistent with Pickering's conceptualization of masked hypertension.³² Whereas persons with white coat hypertension may have conditional-anxiety during the medical visit because of a history of elevated office BP readings, individuals with masked hypertension are less likely to have been labeled with hypertension, and present in a low-anxiety state during their office-based BP assessment.^{33,34}

The masked hypertension phenotype comprises a unique group of young women who are undetected in routine clinical settings. They are at increased risk of future cardiovascular events and end/target organ damage.^{9–11} An analysis of data harmonized from 5 population studies of HBPM found that participants with untreated masked hypertension had higher cardiovascular and mortality risk than individuals with normal BP (hazard ratio [HR] 1.55 [1.12, 2.14] and HR 1.36 [1.04, 1.77], respectively) and white coat hypertension (HR 1.42 [1.06, 1.91] and HR 1.36 [1.04, 1.77], HR 1.13 [0.87, 1.46], respectively).⁹ With normal office BPs, and no other abnormal CVD indicators (e.g., obesity), women with masked hypertension are unlikely to receive treatment, partly explaining the associated poor cardiovascular outcomes.¹¹

Table 2. Associations of adverse pregnancy outcomes, neighborhood deprivation, and individual factors with hypertension phenotypes

Masked hypertension (n = 37)			White coat hypertension (n = 37)			Sustained hypertension (n = 62)			
Model 1 aOR (95% CI)	P value	Model 2 aOR (95% CI)	P value	Model 1 aOR (95% CI)	P value	Model 2 aOR (95% CI)	P value	Model 2 aOR (95% CI)	P value
<i>Adverse pregnancy outcomes</i>									
HDP vs. no APO 2.26 (0.85, 6.04)	0.103	1.74 (0.62, 4.90)	0.291	4.88 (2.11, 11.32)	0.000	4.20 (1.66, 10.60)	0.002	5.08 (2.46, 10.49)	<0.001
PTB vs. no APO 1.09 (0.37, 3.22)	0.882	0.81 (0.26, 2.53)	0.720	1.12 (0.39, 3.22)	0.841	1.30 (0.43, 3.96)	0.642	1.47 (0.64, 3.38)	0.362 (0.67, 4.04)
SGA vs. no APO 2.70 (0.88, 8.31)	0.084	1.75 (0.53, 5.73)	0.359	0.62 (0.08, 5.06)	0.655	0.90 (0.10, 7.77)	0.923	2.38 (0.86, 6.63)	3.38 (1.10, 10.38)
Neighborhood deprivation Overall ranking 4.28 (1.24, 14.74)	0.021	1.33 (0.27, 6.50)	0.725	0.57 (0.17, 1.96)	0.374	0.74 (0.14, 3.89)	0.722	2.15 (0.82, 5.64)	0.119 (0.36, 4.76)
<i>Individual factors</i>									
Perceived stress Total score 1.16 (1.04, 1.29)	0.006	1.15 (1.03, 1.28)	0.014	1.04 (0.92, 1.16)	0.559	1.00 (0.89, 1.13)	0.981	1.07 (0.98, 1.17)	0.141 (0.96, 1.16)
Age in years				0.489		1.07 (0.99, 1.16)	0.088		
Race is Black				0.131		0.55 (0.16, 1.91)	0.342		
College educated				0.191		0.62 (0.24, 1.56)	0.307		
Visit BMI (kg/m ²)				0.804		1.09 (1.04, 1.14)	0.001		

Abbreviations: aOR, adjusted odds ratio; APO, adverse pregnancy outcome; BMI, body mass index; CI, confidence interval; HDP, hypertension disorder of pregnancy; PTB, preterm birth; SGA, small for gestation age infant. APO categories are mutually exclusive, bolded = P < 0.05.

HBPM has the advantage of capturing ecologically valid individual differences in BP, thus reflecting the impact of environmental stressors. This is aligned with our finding that higher perceived stress and neighborhood-level deprivation were only associated with masked hypertension. Masked hypertension has been studied extensively in occupational settings, with clear linkages to job strain, and recent work showing an association with long working hours.^{34–36} Our finding that a greater proportion of young Black women had masked hypertension than women of other racial identities is consistent with other research.^{13,37} Black women are overrepresented in lower socio-economic jobs, which correlates with living in economically deprived neighborhoods, which is consistent with the evidence linking job strain and neighborhood stress with masked hypertension. Based on Pickering's conditional-anxiety hypothesis of masked hypertension and the research on racism and health, we speculate that there are many occurrences in the daily lives of young Black women where a conditioned anxiety response occurs, such as in response to repeated microaggressions or other forms of racism.³⁸ Frequent conditioned-anxiety responses and related HPA axis dysfunction could partly explain the worse outcomes for masked hypertension compared with sustained or white coat hypertension phenotypes. This testable hypothesis aligns with the health deterioration associated with chronic stress (e.g., allostatic load, weathering) and requires further exploration.³⁹

The findings are to be interpreted in balance with some potential limitations. Regarding neighborhood deprivation, data were unavailable on resilience factors such as neighborhood cohesiveness which may buffer the challenges of living in a neighborhood with greater deprivation. Similarly, the time participants resided at their address is unknown. Also, we have limited power to detect some associations due to the relatively small number of women within each hypertension phenotype. Thus, the null findings are interpreted as preliminary and require further study in larger, clinically diverse samples. The parent study required that the placenta from the index pregnancy be sent for examination.⁵ As a result, our sample likely includes a greater proportion of women with pregnancy complications, which may conservatively bias our findings. However, the high proportion of women with APO is a strength of the current study and allowed us to evaluate the association of hypertension phenotypes with PTB and SGA, in addition to HDP. Further study using ambulatory BP monitoring could identify other phenotypes, including nocturnal hypertension, beyond the first year postpartum and examine underlying vascular dysfunction and other mechanisms.

The current study contributes to a growing body of literature on the prevention of CVD after APO. Early detection and treatment of CVD risk factors are a key feature of CVD prevention and should include regular BP monitoring. Currently, HBPM is recommended for white coat hypertension and the guidelines indicate it is reasonable when office BP is high normal to screen for masked hypertension.^{21,40} In addition, a cost-benefit analysis showed cost savings for HBPM as early as age 20; however, it is not currently reimbursable by many insurance carriers to establish the diagnosis of hypertension.⁴¹ Our findings suggest additional research and policy are needed to expand the evidence base and insurer support for home or ambulatory monitoring for women with high normal BP in the office setting, a history of APO, or high psychosocial stress.^{37,40,42} Out-of-office BP monitoring could be an important component of future interventions to mitigate the risk of CVD after an APO.

SUPPLEMENTARY MATERIAL

Supplementary materials are available at *American Journal of Hypertension* (<http://ajh.oxfordjournals.org>).

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CONFLICT OF INTEREST

Esa M. Davis is a member of the US Preventive Services Tasks Force (USPSTF). This article does not necessarily represent the views and policies of the USPSTF.

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