Version of Record at: https://doi.org/10.1111/jch.14856

Received: 15 March 2024 | Revised: 1 May 2024 | Accepted: 17 May 2024

DOI: 10.1111/jch.14856

ORIGINAL ARTICLE

WILEY

Device-estimated sleep metrics do not mediate the relation between race and blood pressure dipping in young black and white women

Michele N. D'agata MS[1] | Elissa K. Hoopes PhD[2] | Thomas Keiser BS[2] | Freda Patterson PhD[2] | Krista M. Szymanski MS[1] | Alexs A. Matias MS[1] | Benjamin C. Brewer MS[3] | Melissa A. Witman PhD[1]

[1]Department of Kinesiology and Applied Physiology, College of Health Sciences, University of Delaware, Newark, Delaware, USA

[2]Department of Health Behavior and Nutrition Sciences, College of Health Sciences, University of Delaware, Newark, Delaware, USA

[3]Department of Epidemiology, College of Health Sciences, University of Delaware, Newark, Delaware, USA

Correspondence
Melissa A. Witman, PhD, Department of Kinesiology and Applied Physiology, College of Health Sciences, University of Delaware, Newark, DE, USA.
Email: mwitman@udel.edu

Funding information
University of Delaware Research Foundation (UDRF); American Heart Association, Grant/Award Number: 831488; University of Delaware Graduate College Doctoral Fellowship for Excellence

Abstract

Short, disturbed, and irregular sleep may contribute to blunted nocturnal blood pressure (BP) dipping, a predictor of cardiovascular disease. Black women (BLW) demonstrate less BP dipping and poorer sleep health than White women (WHW). However, it remains unclear whether device-estimated sleep health metrics mediate the relation between race and BP dipping in young women. We hypothesized that the relation between race and BP dipping would be partly mediated by sleep health metrics of sleep duration, sleep efficiency, and sleep regularity. Participants (20 BLW, 17 WHW) were 18–29 years old, normotensive, nonobese, and without evidence of sleep disorders. Systolic and diastolic BP dipping were derived from 24-h ambulatory BP monitoring. Habitual sleep duration and sleep efficiency were estimated via 14 days of wrist actigraphy. Sleep duration regularity was calculated as the standard deviation (SD) of nightly sleep duration (SDSD). Sleep timing regularity metrics were calculated as the SD of sleep onset and sleep midpoint (SMSD). Mediation analysis tested the mediating effect of each sleep metric on the relation between race and BP dipping. BLW experienced less systolic ($P = .02$) and diastolic ($P = .01$) BP dipping. Sleep duration ($P = .14$) was not different between groups. BLW had lower sleep efficiency ($P < .01$) and higher SDSD ($P = .02$), sleep onset SD ($P < .01$) and SMSD ($P = .01$). No sleep metrics mediated the relation between race and BP dipping (all indirect effects $P > .38$). In conclusion, mediation pathways of sleep health metrics do not explain racial differences in nocturnal BP dipping between young BLW and WHW.

1 | INTRODUCTION

Cardiovascular diseases (CVD) disproportionately affect non-Hispanic Black women (BLW) compared with women of other races and ethnicities in the U.S.[1] Hypertension (HTN) is one of the strongest independent predictors of CVD and is highly prevalent among young BLW, with evidence indicating that BLW aged 18–35 years have a 58% higher likelihood of having HTN compared with White women (WHW).[2] Accordingly, a recent report from the Study of Women's Health Across the Nation suggests that blood pressure (BP)-related

interventions for BLW should be initiated as early as 30 years of age due to the early onset of HTN in this population.[3]

In addition to daytime BP, racial differences in nocturnal BP and BP dipping have also been reported between BLW and WHW.[4] Elevated nocturnal BP is an independent predictor of CVD that may be more sensitive at predicting cardiovascular outcomes than daytime BP,[5] and the presence of non-dipping BP (<10% reduction in nighttime BP from average daytime values) further exacerbates CVD risk.[6] Specifically, non-dipping BP is associated with excess CVD risk from cardiac damage, HTN-mediated organ damage, and altered nocturnal sympathetic tone[5] and every 5% attenuation in nocturnal BP dipping is associated with a 20% increased risk for cardiovascular mortality.[5] Importantly, non-dipping BP may occur independent of elevated nocturnal BP, making this an early independent marker of CVD risk.[5] Previous studies conducted in young and middle-aged adults demonstrate that the prevalence of non-dipping BP is higher among BLW compared with WHW, independent of clinic or daytime BP and other health factors including physical activity, body mass index (BMI), or a single night of sleep.[7–9] Thus, the contributors to the observed racial differences in nocturnal BP dipping between young BLW and WHW remain to be fully elucidated.

Short and disturbed sleep are independent risk factors for CVD and more recently, irregular sleep duration and timing (i.e., inconsistent day-to-day sleep duration and sleep-wake times) have been reported to independently associate with CVD development.[10,11] Racial differences in sleep health have also been identified indicating shorter sleep duration[12] and lower sleep efficiency[7] among young BLW compared with WHW, and more irregular sleep duration and timing[13] among Black compared with White older adults. Habitually short and irregular sleep patterns are linked to non-dipping BP via disturbances in autonomic balance and circadian misalignment, which subsequently disturb 24-h BP patterns.[14–16] It has also been reported that actigraphy-derived estimates of sleep efficiency over seven nights are positively associated with nocturnal systolic BP dipping in young adults.[17] Thus, racial differences in metrics of habitual sleep health identified early in life have been proposed as contributors to the higher prevalence of non-dipping BP among Black adults.[18]

One study has explored the influence of a single night of sleep on racial differences in nocturnal BP dipping between young, otherwise healthy Black and White adults,[7] though none have explored the influence of device-estimated habitual sleep duration and sleep efficiency, and only one recent study has explored the influence of sleep regularity.[16] Given that racial differences in sleep health and BP dipping emerge during early adulthood,[7,9] this is a critical time point for investigation. Further, cumulative evidence indicates that women exhibit greater vulnerability to the adverse cardiovascular effects of poor sleep health, including disruptions in nocturnal BP, as compared with men.[19,20]

Therefore, the purpose of this study was to examine the influence of device-estimated sleep health metrics (sleep duration, sleep efficiency, and sleep regularity) on racial differences in nocturnal BP dipping between apparently healthy, young adult BLW and WHW. We hypothesized that BLW would have lower nocturnal systolic and diastolic BP dipping, as well as shorter sleep duration, lower sleep efficiency, more irregular sleep duration, and more irregular sleep timing compared with WHW. We further hypothesized that sleep metrics would at least partly meditate the relation between race and nocturnal BP dipping.

2 | METHODS

2.1 | Study participants and protocol

Data used in this study are from two separate protocols within the same research laboratory approved by the Institutional Review Board at the University of Delaware (IRB Study No. 1704969 and 1957713). Data from IRB Study No. 1957713 includes baseline data from a larger clinical trial that is registered on ClinicalTrials.gov (ID: NCT05656742). Each study was conducted in accordance with the ethical standards of the *Declaration of Helsinki* and written informed consent was obtained from all participants. Participants were recruited from the University of Delaware and the surrounding Newark, DE region and included apparently healthy cisgender women between the ages of 18 and 29 years who self-identified their ethnicity as non-Hispanic and self-identified their race as Black or White. Participants were non-shift working, free of sleep disorders or conditions known to affect sleep (e.g., depression), not taking medication or supplements known to affect sleep, non-smoking, had a BMI $\leq$ 30 kg/m^2, and had screening seated resting BP < 140 and < 90 mmHg. Participants were excluded if they had a history of chronic diseases or conditions, including cardiovascular, renal, metabolic, autoimmune, cancerous conditions, or a recent history of coronavirus disease 2019 infection (<60 days) or vaccination (<14 days). Participants were premenopausal, not pregnant, and not currently breastfeeding. All but one participant (n = 1 WHW) were nulliparous. Nineteen women were naturally cycling (13 BLW, 6 WHW) and 18 women reported currently using hormonal birth control (oral: 5 BLW, 6 WHW; hormonal IUD: 1 BLW, 5 WHW; hormonal implant: 1 BLW). However, menstrual cycle was not controlled throughout the protocol.

After consenting, screening procedures required participants to complete a brief review of medical history and were asked to report on current medication use, menstrual history and hormonal contraceptive use, weekly caffeine intake reported in number of drinks per week, educational attainment, and parent history of HTN. Participants also underwent screening for the presence of high risk of insomnia (Insomnia Severity Index score $\geq$ 15)[21] and sleep apnea (STOP-bang score $\geq$ 3).[22] Seated resting BP was assessed to confirm screening BP was < 140 and < 90 mmHg (Omron 5 Series, BP7200). Height and weight were measured for the calculation of BMI and body fat percentage was determined via bioelectrical impedance analysis (Tanita TBF-300A, Arlington Heights, IL). Eligible participants were equipped with an accelerometer (Actiwatch Spectrum Plus; Philips-Respironics, Inc.) that they were instructed to wear on their non-dominant wrist for 14 consecutive days and nights for estimation of sleep metrics and physical activity. Participants were also equipped with an ambulatory BP monitor (ABPM) which they were instructed to wear for a single 24-

h period either during or within 1-week after sleep monitoring. After completion of sleep monitoring (day 15), participants were instructed to report to the laboratory during the morning hours (between 07:00 and 11:30), following an overnight fast, without caffeine for ≥ 12 h, without alcohol, exercise, or vitamins/supplements for ≥ 24, and without over-the-counter medications or anti-inflammatory drugs for ≥ 72 h prior to the visit. Upon arrival at the laboratory, intravenous blood sampling was performed for the clinical assessment of fasting blood glucose and a lipid panel (Study No. 1704969: Quest Diagnostics, Inc., Philadelphia, PA; Study No. 1957713: LabCorp Testing Services).

2.2 | Device-estimated sleep metrics

Sleep metrics were estimated via wrist actigraphy (Actiwatch Spectrum Plus) as previously described.[23] In short, participants were instructed to wear the Actiwatch continuously on their non-dominant wrist for 14 days and nights, in conjunction with a standardized daily sleep diary to assist with sleep-wake scoring.[24] To estimate habitual sleep health metrics, a conservative minimum of 10 days and nights of wear was required for inclusion in analyses.[25] Data were collected in 30-second epochs and processed by trained investigators (M.N.D., E.K.H., and T.K.) using both participants' standardized sleep diaries and Philips Actiware software (version 6.1.0). Nights with > 1 h of missing data were considered invalid and daytime naps were excluded from nighttime sleep scoring.[26] Rest intervals were identified using a standardized protocol that incorporates sleep diaries, activity levels, and "lights out."[26,27] Sleep-wake scoring was based on the medium threshold setting for sleep/wake detection using the algorithm provided by the manufacturer. For each night of wear, sleep duration was estimated as the total time scored as sleep between sleep onset and sleep offset. Sleep efficiency was calculated as total sleep time divided by total time in bed dedicated to sleep, expressed as a percentage. Mean values were generated to characterize habitual sleep duration and sleep efficiency for each participant over the 14-day wear period. Sleep duration regularity was calculated as the standard deviation (SD) of nightly sleep duration (SDSD). Sleep timing was operationalized as the timing of sleep onset (clock time at start of each nocturnal sleep period) and the timing of sleep midpoint (clock time halfway between sleep onset and sleep offset). Sleep timing regularity was calculated as the SD of sleep onset timing (sleep onset SD) and the SD of sleep midpoint timing (SMSD). Two BLW were excluded from sleep timing regularity analyses due to the fact that their sleep monitoring spanned daylight savings time change (March 12, 2023). For all other sleep metrics, sleep the night of daylight savings was excluded from analyses for those participants.

2.3 | Ambulatory BP monitoring

Participants were fitted with a calibrated ABPM (Oscar 2; SunTech Medical Inc, Morrisville, NC) to be worn for 24 h. ABPM was conducted during or within 1-week after the 14-day sleep monitoring period. Most participants (15 BLW, 16 WHW) wore the Actiwatch during the ABPM period, thus acute sleep duration and sleep efficiency on the night of ABPM were assessed for this large subset of participants. Participants were provided with detailed instructions regarding the use of the ABPM and were asked to refrain from planned exercise, alcohol, over-the-counter medications/anti-inflammatory drugs, and excessive caffeine (i.e., > 1 cup of coffee) during the 24-h wear period. Monitors were pre-programmed to automatically record brachial systolic and diastolic BP every 20 min during estimated waking hours and every 30 min during estimated sleeping hours. For inclusion in analyses, a minimum of 14 daytime and seven nighttime BP readings were required.[28] Participants were also asked to record their sleep and wake times on the night of ABPM, which was used to retrospectively adjust the pre-programmed sleep-wake period for accurate calculation of daytime and nighttime BP. Daytime systolic and diastolic BP were each calculated as the average of the readings obtained from the awake period and nighttime systolic and diastolic BP were each calculated as the average of the readings obtained from the sleep period. Systolic and diastolic BP dipping (%) were then each calculated as [(wake BP—sleep BP)/wake BP)] × 100. For descriptive purposes, non-dipping BP was defined as < 10% reduction in BP from wake to sleep and dipping BP was defined as ≥ 10% reduction in BP from wake to sleep.[29] Heart rate (HR) was also obtained from 24-h ABPM and HR dipping (%) was calculated as [(wake HR—sleep HR)/wake BP)] × 100.

2.4 | Physical activity estimates

To provide an estimate of daily physical activity, average daily total activity counts (TAC) and average activity counts per minute (CPM) were derived from the Actiware Software. Average daily TAC is a global measure of physical activity captured by accelerometers and a proxy for the total volume of physical activity performed.[30] Rest intervals and days with < 10 h of wear time were excluded from analyses.[31] TAC and CPM the day before ABPM were also derived in a subset of participants who had a full day of wear prior to the start of ABPM ($n = 29$; 13 BLW, 16 WHW) to test if differences in physical activity the day before ABPM may contribute to differences in 24-h BP patterns.[32]

2.5 | Covariates

Age, BMI, parent history of HTN, weekly caffeine consumption, respective daytime BP, daytime HR, nighttime HR, and HR dipping% were tested as potential covariates. Age, BMI, parent history of HTN, and weekly caffeine consumption were obtained at screening. Daytime systolic and diastolic BP, daytime HR, nighttime HR, and HR dipping% were obtained during ABPM. These covariates were selected on a theoretical basis as each of these variables have an established association with BP[33–37] or sleep[38,39] and thus may influence the interrelatedness of these variables.

2.6 | Statistical analyses

Summary statistics were used to characterize participants within each group (BLW vs. WHW). Normality was confirmed for nocturnal BP dipping and sleep metrics using Shapiro-Wilk tests of normality and thus parametric statistical tests were used. Group differences in continuous variables for participant characteristics, 24-h BP metrics, and sleep metrics were analyzed using independent samples *t*-tests and effect sizes were calculated using Cohen's *d*. Group differences in categorical variables were evaluated using Fisher's exact test. Separate linear regression models of BP dipping were first used to establish race as an independent predictor of both systolic and diastolic nocturnal BP dipping in the absence of sleep metrics using age, BMI, parent history of HTN, weekly caffeine consumption, respective daytime BP, daytime HR, nighttime HR, and HR dipping% as covariates. Separate linear regression models of BP dipping were also performed to test independent associations between sleep metrics and BP dipping, irrespective of race. The standard mediation model as proposed by Baron and Kenney in 1986[40] was used to assess the mediating effect of habitual sleep duration, sleep efficiency, and SDSD on the relation between race and systolic and diastolic BP dipping. We also conducted exploratory analyses in a subset of participants (15 BLW, 16 WHW) to evaluate the mediating effect of acute sleep duration and sleep efficiency the night of ABPM on the relation between race and systolic and diastolic BP dipping. Fisher's exact test was performed with GraphPad Prism Software (v 10.1.2, San Diego, CA) and all other analyses was performed with *jamovi* (Version 2.3.26) [Computer Software], Sydney, Australia. Statistical significance was set a priori at $\alpha \leq 0.05$. Summary statistics are presented as means ± SD or *n* (%) for continuous and categorical variables, respectively.

3 | RESULTS

3.1 | Participants

Fifty participants (26 BLW, 24 WHW) were screened for eligibility, of which 10 participants (3 BLW, 7 WHW) were deemed ineligible for participation due to not meeting at least 1 of the screening criteria and three participants (3 BLW, 0 WHW) withdrew from the study prior to completion. Thus, 37 participants (20 BLW, 17 WHW) completed the study and were included in final analyses. Participant characteristics for each group are displayed in Table 1. All participants were young (18-29 years), apparently healthy women. WHW had significantly higher fasting blood glucose, reported consuming more caffeine, and had fewer reports of parent history of HTN. Daytime and nighttime ambulatory BPs were not different between groups ($P > .16$), although WHW had significantly lower daytime ($P = .05$) and nighttime HR ($P < .01$) and greater nighttime HR dipping ($P = .05$) compared with BLW (Table 1).

Physical activity assessed as average daily TAC was not different between groups (BLW: 230468 ± 55154, WHW: 230357 ± 47812; $P = .99$). Average activity CPM was also not different between groups (BLW: 260 ± 64, WHW: 249 ± 47; $P = .58$). Average daily accelerometer wear time was not different between groups (BLW: 15.7 ± 0.7 h, WHW: 15.9 ± 0.9 h; $P = .32$). In addition, TAC (BLW: 219602 ± 88323, WHW: 237010 ± 55497; $P = .52$) and CPM (BLW: 253 ± 118, WHW: 246 ± 47; $P = .84$) were not different between groups the day before ABPM.

3.2 | Racial differences in nocturnal BP dipping and sleep metrics

Figure 1 compares nocturnal BP dipping between races and demonstrates that BLW had significantly less systolic ($P = .02$) and diastolic ($P = .01$) BP dipping compared with WHW. There was also a large effect of race on nocturnal systolic BP dipping ($d = 0.80$) and nocturnal diastolic BP dipping ($d = 0.87$). BLW also experienced less HR dipping than WHW (Table 1). Though not significant, more BLW were classified as non-dippers than WHW (Table 1). Racial differences in habitual sleep metrics were also detected such that BLW had significantly lower sleep efficiency ($P < .01$) as well as significantly higher SDSD ($P = .02$), sleep onset SD ($P < .01$), and SMSD ($P = .01$) compared with WHW, however, sleep duration was not different between groups ($P = .14$) (Table 1). Sleep efficiency on the night of ABPM was significantly lower among BLW ($P < .01$) while sleep duration was not different between groups ($P = .79$) (Table 1).

3.3 | Testing covariates

Linear regression models of BP dipping were used to establish race as an independent predictor of nocturnal systolic and diastolic BP dipping. Race remained a significant predictor of nocturnal systolic BP dipping in separate models that adjusted for age (Estimate: −5.94, SE: 2.01, $P < .01$; years), BMI (Estimate: − 4.44, SE: 1.85, $P = .02$; kg/m^2), parent history of HTN (Estimate: − 7.31, SE: 1.89, $P < .001$; yes), caffeine consumption (Estimate: − 4.47, SE: 2.01, $P = .03$; drinks per week), daytime systolic BP (Estimate: − 4.25, SE: 1.84, $P = .03$; mmHg), daytime HR (Estimate: − 5.29, SE: 1.91, $P < .01$; beats per minute), nighttime HR (Estimate: − 6.29, SE: 2.00, $P < .01$; beats per minute), and HR dipping% (Estimate: − 5.10, SE: 1.93, $P = .01$; %). Similarly, race remained a significant predictor of nocturnal diastolic BP dipping after adjustment age (Estimate: − 7.48, SE: 2.36, $P < .01$; years), BMI (Estimate: − 5.60, SE: 2.18, $P = .02$; kg/m^2), parent history of HTN (Estimate: − 8.46, SE: 2.31, $P < .001$; yes), caffeine consumption (Estimate: − 5.70, SE: 2.37, $P = .02$; drinks per week), daytime systolic BP (Estimate: − 5.08, SE: 2.13, $P = .02$; mmHg), daytime HR (Estimate: − 6.95, SE: 2.19, $P < .01$; beats per minute), nighttime HR (Estimate: − 8.02, SE: 2.33, $P < .01$; beats per minute), and HR dipping% (Estimate: − 6.16, SE: 2.29, $P = .01$; %).

TABLE 1 Group participant characteristics.

Participant characteristics	BLW	WHW	Cohen's *d*	*P*-value
n	20	17	–	–
Age, years	22 ± 3	25 ± 3	1.00	**<.01**
BMI, kg/m^2	23.6 ± 3.6	23.7 ± 2.8	0.03	.94
Total-C, mg/dL	171 ± 31	169 ± 27	0.07	.89
LDL-C, mg/dL	94 ± 31	91 ± 18	0.12	.72
HDL-C, mg/dL	65 ± 13	62 ± 13	0.23	.59
Glucose, mg/dL	83 ± 5	89 ± 5	1.20	**<.01**
Caffeine consumption, drinks/week	4 ± 6	8 ± 4	0.78	**.02**
Parent history of HTN, *n* (%)	12 (60)	2 (12)	–	**.01**
Education, *n* (%)				
Formal Education post High School	20 (100)	17 (100)	–	>.99
College Graduate	13 (65)	14 (82)	–	.29
Post-Graduate Education	7 (35)	6 (35)	–	>.99
24-h ABPM	**BLW**	**WHW**		***P*-value**
Daytime SBP, mmHg	120 ± 10	122 ± 14	0.16	.53
Daytime DBP, mmHg	69 ± 8	70 ± 7	0.13	.53
Daytime HR, bpm	79 ± 10	72 ± 12	0.63	**.05**
Nighttime SBP, mmHg	108 ± 9	105 ± 14	0.25	.47
Nighttime DBP, mmHg	57 ± 6	55 ± 5	0.36	.16
Nighttime HR, bpm	71 ± 12	60 ± 9	1.03	**<.01**
HR dip, %	10 ± 9	16 ± 8	0.70	**.05**
SBP non-dippers, *n* (%)	9 (45)	3 (18)	–	.09
DBP non-dippers, *n* (%)	4 (20)	0 (0)	–	.11
Habitual sleep	**BLW**	**WHW**		***P*-value**
Sleep duration, hours	6.7 ± 0.5	7.0 ± 0.7	0.49	.14
Sleep efficiency, %	81.6 ± 4.4	86.0 ± 3.1	1.16	**<.01**
SDSD, min	77 ± 22	61 ± 24	0.69	**.02**
[a]Sleep Onset SD, min	76 ± 25	46 ± 18	1.38	**<.01**
[a]SMSD, min	58 ± 19	42 ± 15	0.93	**.01**
Acute sleep (During ABPM)	**BLW**	**WHW**		***P*-value**
[b]Sleep duration, hours	7.2 ± 1.4	7.1 ± 0.9	0.08	.79
[b]Sleep efficiency, %	82.1 ± 5.1	87.1 ± 4.9	1.00	**<.01**

Notes: Values presented as means ± standard deviation for continuous variables or *n* (%) for categorical variables. Independent samples *t-tests* were used to assess group differences for continuous variables and Fisher's exact tests were used to assess group differences in categorical variables ($n = 37$; 20 BLW and 17 WHW). Cohen's d was used to examine effect sizes between groups for continuous variables. *P*-values in bold indicate significance set a priori at $\alpha \leq 0.05$. Abbreviations: BLW, Black women; BMI, body mass index; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; HR, heart rate; HTN, hypertension; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; SDSD, sleep duration standard deviation; Total-C, total cholesterol; WHW, White women.

[a]Sleep timing regularity metrics were calculated in a subset of participants ($n = 35$; 18 BLW, 17 WHW).

[b]Acute sleep metrics during the night of ABPM were obtained in a subset of participants ($n = 31$, 15 BLW and 16 WHW).

3.4 | No associations between habitual sleep metrics and nocturnal BP dipping

In pooled analyses no habitual sleep metrics were associated with nocturnal systolic BP dipping (sleep duration [Estimate: 2.36, SE: 1.56, $P = .14$; h]; sleep efficiency [Estimate: 0.29, SE: 0.22, $P = .20$; %]; SDSD [Estimate: – 0.02, SE: 0.04, $P = .56$; min]; sleep onset SD [Estimate: – 0.06, SE: 0.04, $P = .12$; min]; SMSD [Estimate: – 0.07, SE: 0.05, $P = .22$; min]). Similarly, in pooled analyses, no habitual sleep metrics were associated with nocturnal diastolic BP dipping (sleep duration [Estimate: 2.65, SE: 1.87, $P = .17$; h]; sleep efficiency [Estimate: 0.23, SE: 0.27, $P = .40$; %]; SDSD [Estimate: – 0.04, SE: 0.05, $P = .42$; min]; sleep onset

Version of Record at: https://doi.org/10.1111/jch.14856

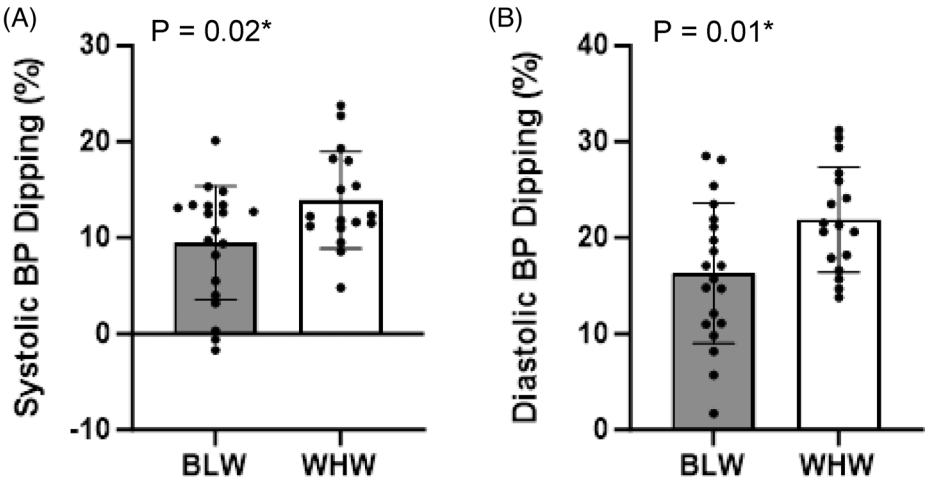

FIGURE 1 Racial differences in nocturnal BP dipping. Nocturnal systolic (A) and diastolic (B) BP dipping (%) are shown for Black women (BLW, $n = 20$) and White women (WHW, $n = 17$). Independent samples *t*-tests were used to assess group differences in nocturnal BP dipping. BP, blood pressure. * indicates significance set a priori at $\alpha \leq 0.05$.

SD [Estimate: – 0.07, SE: 0.05, $P = .14$; min]; SMSD [Estimate: – 0.08, SE: 0.07, $P = .23$; min]).

3.5 | No mediating effect of sleep health metrics on racial differences in nocturnal BP dipping

In contrast to our hypothesis, no habitual sleep metrics were found to mediate the relation between race and nocturnal systolic BP dipping (Table 2) or the relation between race and nocturnal diastolic BP dipping (Table 3). Specifically, race was a significant predictor of both systolic and diastolic BP dipping in both the absence (*total effect*) and the presence (*direct effect*) of each mediator (sleep duration, sleep efficiency, SDSD, sleep onset SD, and SMSD). Further, no indirect effect of race on nocturnal BP dipping was found, indicating no mediating effect of habitual sleep metrics on this direct relation. Race was a significant predictor of nocturnal HR dipping ($P = .05$), but no sleep metrics mediated this association (*data not shown*).

Neither sleep duration nor sleep efficiency on the night of ABPM were found to mediate the relation between race and nocturnal systolic and diastolic BP dipping. When sleep duration was the hypothesized mediator, no significant indirect effect of race on nocturnal systolic ($P = .79$) or diastolic ($P = .96$) BP dipping was found. Similarly, when sleep efficiency was the hypothesized mediator, no significant indirect effect of race on nocturnal systolic ($P = .93$) or diastolic ($P = .63$) BP dipping was found.

4 | DISCUSSION

To our knowledge, this is the first study to evaluate the effect of device-estimated habitual sleep health metrics on racial differences in nocturnal BP dipping between young BLW and WHW. Findings from this study indicate less systolic and diastolic nocturnal BP dipping among young, otherwise healthy BLW compared with WHW. Racial differences in nocturnal BP dipping were accompanied by lower sleep efficiency as well as increased SDSD, sleep onset SD, and SMSD among BLW compared with WHW. However, in contrast to our hypothesis, these observed differences in habitual sleep health metrics did not mediate the association between race and nocturnal BP dipping. Further, we did not find any mediating effect of acute sleep duration or sleep efficiency the night of ABPM on the relation between race and nocturnal BP dipping.

Sleep is a proposed contributor to observed racial differences in nocturnal BP dipping based on evidence of worse sleep health in Black compared with White individuals across the lifespan, in combination with evidence linking short and disturbed sleep to impairments in nocturnal BP dipping.[18] Still, few studies have attempted to evaluate the influence of sleep on racial differences in nocturnal BP dipping. Findings from this study align with a previous study by Hughes and colleagues[7] that reported shorter sleep duration and lower sleep efficiency as well as less systolic and diastolic BP dipping among normotensive young adult BLW compared with WHW, but did not find that racial differences in sleep accounted for BP dipping differences.[7] Similar to the present study, HR dipping was also significantly lower among BLW compared with WHW but neither sleep duration nor sleep efficiency explained racial differences in HR dipping. However, participants included in that study only completed a single night of wrist actigraphy on the night of ambulatory BP monitoring, limiting conclusions to be drawn regarding the influence of habitual sleep duration and sleep efficiency as well as the influence of sleep regularity on racial differences in BP dipping.[7]

Conversely, our findings from both pooled analyses and analyses separated by race oppose two previous investigations conducted in middle-aged adults with HTN.[41,42] Specifically, of the several sleep health metrics we tested, none were found to be associated with nocturnal BP dipping in our sample of young non-hypertensive women,

TABLE 2 Mediating effect of habitual sleep metrics on the relation between race and nocturnal systolic BP dipping.

			95% confidence interval				
Effect	**Estimate**	**SE**	**Lower**	**Upper**	**Z**	***P*-value**	**% Mediation**
A. Sleep duration							
Indirect	-0.47	0.54	-1.53	0.59	-0.87	.382	10.63
Direct	-3.97	1.81	-7.51	-0.43	-2.20	**.028**	89.37
Total	-4.44	1.78	-7.92	-0.96	-2.50	**.012**	100.00
B. Sleep efficiency							
Indirect	-0.17	1.04	-2.22	1.88	-0.16	.871	3.83
Direct	-4.27	2.06	-8.31	-0.23	-2.07	**.038**	96.17
Total	-4.44	1.78	-7.92	-0.96	-2.50	**.012**	100.00
C. Sleep duration standard deviation							
Indirect	0.23	0.73	-1.21	1.67	0.32	.752	4.71
Direct	-4.67	1.92	-8.43	-0.92	-2.44	**.015**	95.29
Total	-4.44	1.78	-7.92	-0.96	-2.50	**.012**	100.00
D. Sleep onset standard deviation							
Indirect	-0.40	1.28	-2.92	2.12	-0.31	.757	8.25
Direct	-4.43	2.24	-8.82	-0.04	-1.98	**.048**	91.75
Total	-4.83	1.84	-8.43	-1.23	-2.63	**.009**	100.00
E. Sleep midpoint standard deviation							
Indirect	-0.24	0.90	-2.00	1.53	-0.26	.793	4.90
Direct	-4.59	2.04	-8.60	-0.59	-2.25	**.025**	95.10
Total	-4.83	1.84	-8.43	-1.23	-2.63	**.009**	100.00

Note: Standard mediation was used to determine the indirect effect of sleep duration (A), sleep efficiency (B), sleep duration standard deviation (C), sleep onset standard deviation (D), and sleep midpoint standard deviation (E) on the relation between race and nocturnal systolic BP dipping. The effect of race on systolic BP dipping in the presence (*direct effect*) and absence (*total effect*) of the meditating effect of habitual sleep variables are also shown. *P*-values in bold indicate significance set a priori at $\alpha \leq 0.05$.

irrespective of race. Comparatively, using nine consecutive nights of wrist actigraphy and 48-h of ambulatory BP monitoring, Matthews and colleagues[41] reported that shorter sleep duration and greater sleep fragmentation (i.e., disruption) were associated with less nocturnal systolic and diastolic BP dipping in a sample of Black and White adults, though findings were not stratified by race or sex. Sherwood and colleagues[42] reported that among adults with untreated daytime HTN, Black adults experienced less nocturnal systolic BP dipping and worse sleep quality (assessed from the Pittsburgh Sleep Quality Index and wrist actigraphy during the night of ambulatory BP monitoring) compared with White adults and that sleep quality was positively associated with systolic BP dipping in the whole sample. Further controlling for sleep quality, BMI, and daytime-nighttime difference in urinary catecholamine excretion attenuated racial differences in systolic BP dipping.[42] However, an apparent limitation of the study was the inclusion of smokers and night-shift workers, among which Black adults made up a greater proportion, potentially confounding study findings.[42] Importantly, when compared with findings from Matthews and colleagues[41] and Sherwood and colleagues,[42] lack of association between sleep health metrics and nocturnal BP dipping observed in the present study and in the study by Hughes and colleagues[7] suggests that the effect of poor sleep health on nocturnal BP dipping may not be evident in young adulthood and may instead become more pronounced with age.

In our study, we examined the effect of both habitual and acute device-estimated sleep metrics on racial differences in nocturnal BP dipping in young women. Previous work investigating the effect of sleep on nocturnal BP dipping in young, otherwise healthy Black and White adults has been limited by the lack of inclusion of habitual sleep assessments.[7] Thus, our study is strengthened by the inclusion of understudied sleep metrics in this context, sleep regularity, which has been associated with less nocturnal systolic BP dipping[16] and has been shown to predict CVD risk, independent of traditional CVD risk factors and other sleep metrics.[11] Our findings partly agree with recent findings from Xu and colleagues[16] which reported less nocturnal systolic BP dipping and higher SMSD as assessed by 7 days of wrist actigraphy in Black compared with White young and middle-aged adults. Although in pooled analyses they further report that every 1-h increase in SMSD was associated with a 1.16% decrease in systolic BP dipping, suggesting that racial differences in SMSD may explain racial differences in BP dipping. They also found that every 1-h increase in SDSD was associated with a 1.39% decrease in nighttime systolic BP dipping, however, no racial differences in SDSD were detected between groups and they conclude that SDSD does not explain the observed racial dif-

Version of Record at: https://doi.org/10.1111/jch.14856

TABLE 3 Mediating effect of habitual sleep metrics on the relation between race and nocturnal diastolic BP dipping.

			95% confidence interval				
Effect	**Estimate**	**SE**	**Lower**	**Upper**	**Z**	***P*-value**	**% Mediation**
A. Sleep duration							
Indirect	-0.49	0.61	-1.69	0.72	-0.80	.426	8.73
Direct	-5.11	2.13	-9.29	-0.93	-2.40	**.017**	91.27
Total	-5.60	2.09	-9.70	-1.50	-2.68	**.007**	100.00
B. Sleep efficiency							
Indirect	0.58	1.24	-1.85	3.00	0.47	.641	8.54
Direct	-6.17	2.42	-10.91	-1.44	-2.55	**.011**	91.46
Total	-5.60	2.09	-9.70	-1.50	-2.68	**.007**	100.00
C. Sleep duration standard deviation							
Indirect	0.10	0.86	-1.58	1.78	0.12	.906	1.75
Direct	-5.70	2.26	-10.13	-1.27	-2.52	**.012**	98.25
Total	-5.60	2.09	-9.70	-1.50	-2.68	**.007**	100.00
D. Sleep onset standard deviation							
Indirect	-0.33	1.54	-3.34	2.68	-0.21	.831	5.77
Direct	-5.37	2.68	-10.62	-0.11	-2.00	**.045**	94.23
Total	-5.69	2.20	-10.00	-1.38	-2.59	**.010**	100.00
E. Sleep midpoint standard deviation							
Indirect	-0.24	1.08	-2.34	1.87	-0.22	.825	4.17
Direct	-5.46	2.45	-10.25	-0.66	-2.23	**.026**	95.83
Total	-5.69	2.20	-10.00	-1.38	-2.59	**.010**	100.00

Note: Standard mediation was used to determine the indirect effect of sleep duration (A), sleep efficiency (B), sleep duration standard deviation (C), sleep onset standard deviation (D), and sleep midpoint standard deviation (E) on the relation between race and nocturnal diastolic BP dipping. The effect of race on diastolic BP dipping in the presence (*direct effect*) and absence (*total effect*) of the meditating effect of habitual sleep variables are also shown. *P*-values in bold indicate significance set a priori at $\alpha \leq 0.05$.

ferences in BP dipping.[16] In the present study we did not observe any association between sleep regularity metrics and BP dipping, nor a mediating effect of sleep regularity on racial differences in BP dipping. Participants in the present study were on average 10 years younger and had lower BMI than Xu and colleagues further suggesting that the effects of sleep on nocturnal BP dipping may not become evident until early middle age. This speculation is further supported by prior findings from our laboratory, which indicated that young BLW exhibit lower peripheral microvascular function but preserved macrovascular function when compared with young WHW.[43,44] In a separate investigation we also reported a positive association between SDSD and microvascular function, but not macrovascular function among young adult college students.[45] Collectively, our findings suggest that the cardiovascular consequences of irregular sleep may first manifest as microvascular dysfunction, prior to affecting the macrovasculature and subsequently, BP indices. However, this hypothesis has yet to be systematically tested. Regardless, investigations that attempt to identify modifiable contributors to nocturnal BP dipping during young adulthood are critical as racial differences in nocturnal BP dipping seem to emerge during this time and may underscore the disparate CVD prevalence among Black adults later in life.[9]

This is the first study to report on the effect of sleep on the relation between race and nocturnal BP dipping specifically in young women. The largest disparities in CVD, HTN, and non-dipping BP prevalence occur between BLW and WHW, emerging in early adulthood.[1,9,46] Moreover, young BLWs are more likely to self-report short sleep duration and less likely to report adequate sleep duration as compared with WHW.[12] Interestingly, cumulative evidence indicates that associations between sleep and BP are stronger in women as compared with men.[47] For example, a recent study found that sleep restriction to 4 h per night for 9 days significantly increased 24-h and asleep BP, even after three nights of 9-h recovery sleep in young women but not men, suggesting greater vulnerability to adverse cardiovascular effects of sleep loss in women.[19] Additionally, we have previously reported that increased day-to-day consistency in rest and activity patterns were associated with increased systolic BP dipping in young adult women, but not men, providing support for further investigation of the effect of sleep on racial differences in nocturnal BP dipping exclusively in young women.[20]

4.1 | Experimental considerations

The strengths of this study include employing a robust 14-day sleep monitoring period, while also evaluating the impact of sleep during the night of ABPM on racial differences in nocturnal BP dipping. In healthy

adults, wrist actigraphy-derived sleep health metrics of sleep duration and sleep efficiency have been validated against polysomnography[48] and all sleep variables calculated in the present study have been reported to be highly reproducible, with intra-scorer intraclass correlation coefficients (ICCs) ranging from 0.91 to 0.99.[27] Importantly, the use of multiple consecutive days of wrist actigraphy allowed for the concurrent examination of numerous dimensions of sleep health including sleep duration, sleep efficiency, and sleep regularity. Although not statistically evaluated, our data also demonstrate comparable sleep duration and sleep efficiency during the night of ABPM and over the 14-day monitoring period. During ABPM we used individualized self-report sleep and wake times, rather than fixed sleep-wake intervals, to define daytime and nighttime BP for the calculation of nocturnal BP dipping.[49] This study also included BLW and WHW with similar health profiles as all participants were young (18-29 years), female, non-hypertensive, nonobese, non-smoking, free of sleep disorders, well-educated, and exhibited similar physical activity levels, limiting the influence of these variables on study findings.

We also acknowledge the limitations of this study as acute sleep the night of ABPM was only evaluated in a subset of participants, and we only employed a single night of ABPM. Previous studies suggest that nocturnal BP dipping may vary on a night-to-night basis, thus multiple 24-h periods of ABPM would strengthen study findings.[50] We also did not detect any group differences in daytime or nighttime BP; thus, neither elevated nocturnal BP nor lower daytime BP explained racial differences in BP dipping. Regardless, a non-dipping BP pattern is associated with increased cardiovascular risk, independent of daytime or nighttime BP, making this a clinically relevant marker of cardiovascular health that may precede the development of HTN.[5] Additionally, contributors to racial health disparities are multifaceted and likely extend beyond the variables tested in the present study. We did not assess the influence of other social, behavioral, or physiological determinants of sleep and BP dipping that may differ by race such as daily stress, neighborhood and built environment, or measurement of nighttime melatonin, cortisol, renin, or sympathetic nerve activity, which should be the focus of future investigations.

5 | CONCLUSIONS

Findings from this study support previous work demonstrating attenuated nocturnal BP dipping in BLW compared with WHW. However, we newly found no effect of device-estimated habitual or acute sleep metrics on racial differences in BP dipping in young BLW and WHW. Further, race was the only predictor of nocturnal BP dipping, and in contrast to some previous work, no sleep metrics were found to influence the relation between race and BP dipping. Future work should consider other physiological, social, or behavioral influences of racial differences in nocturnal BP dipping between young, otherwise healthy BLW and WHW to potentially help mitigate the racial disparity in CVD later in life.

AUTHOR CONTRIBUTIONS

M.A.W, F.P, E.K.H, and M.N.D conceived and designed research; M.N.D, E.K.H, T.K, K.M.S, and A.A.M performed experiments; M.N.D, E.K.H, T.K, and B.C.B analyzed data; M.N.D, M.A.W, and B.C.B interpreted results of experiments; M.N.D prepared figures; M.N.D drafted manuscript; All authors edited and revised manuscript; All authors approved final version of manuscript.

ACKNOWLEDGMENTS

The authors thank all the study participants as well as Wendy Nichols, BSN, RN, CCRC, CEN, and the staff at the Nurse Managed Primary Care Center for assistance with blood collections and with the processing of clinical labs for this project. University of Delaware Research Foundation (UDRF)—Strategic Initiative Grant (MAW/FP) and American Heart Association (AHA) Award #831488 (EKH) and the University of Delaware Graduate College Doctoral Fellowship for Excellence.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data contained in the manuscript will be made available upon request.

PATIENT CONSENT STATEMENT

Verbal and written informed consent was obtained from all participants prior to participation.

CLINICAL TRAIL REGISTRATION

ID: NCT05656742 for IRB Study No. 1957713.

PERMISSION TO REPRODUCE MATERIAL FROM OTHER SOURCES

NONE (not applicable).

ORCID

Michele N. D'agata MS https://orcid.org/0000-0002-6166-9627

REFERENCES

1. Tsao CW, Aday AW, Almarzooq ZI, et al. Heart disease and stroke statistics-2022 update: a report from the American Heart Association. *Circulation*. 2022;145:e153-e639.
2. Hines AL, Zare H, Thorpe RJ. Racial disparities in hypertension among young, black and white women. *J Gen Intern Med*. 2022;37:2123-2125. Jr.
3. Reeves A, Elliott MR, Lewis TT, Karvonen-Gutierrez CA, Herman WH, Harlow SD. Study selection bias and racial or ethnic disparities in estimated age at onset of cardiometabolic disease among midlife women in the US. *JAMA Netw Open*. 2022;5:e2240665.
4. Profant J, Dimsdale JE. Race and diurnal blood pressure patterns. A review and meta-analysis. *Hypertension*. 1999;33:1099-1104.
5. Yano Y, Kario K. Nocturnal blood pressure and cardiovascular disease: a review of recent advances. *Hypertens Res*. 2012;35:695-701.
6. Ben-Dov IZ, Kark JD, Ben-Ishay D, Mekler J, Ben-Arie L, Bursztyn M. Predictors of all-cause mortality in clinical ambulatory monitoring: unique aspects of blood pressure during sleep. *Hypertension*. 2007;49:1235-1241.

7. Hughes JW, Kobayashi I, Deichert NT. Ethnic differences in sleep quality accompany ethnic differences in night-time blood pressure dipping. *Am J Hypertens*. 2007;20:1104-1110.
8. James GD. Race and perceived stress independently affect the diurnal variation of blood pressure in women. *Am J Hypertens*. 1991;4:382-384.
9. Booth JN, Anstey DE, Bello NA, et al. Race and sex differences in asleep blood pressure: the Coronary Artery Risk Development in Young Adults (CARDIA) study. *J Clin Hypertens (Greenwich)*. 2019;21:184-192.
10. Buysse DJ. Sleep health: can we define it. *Sleep*. 2014;37:9-17.
11. Huang T, Mariani S, Redline S. Sleep irregularity and risk of cardiovascular events: the multi-ethnic study of atherosclerosis. *J Am Coll Cardiol*. 2020;75:991-999.
12. Amyx M, Xiong X, Xie Y, Buekens P. Racial/ethnic differences in sleep disorders and reporting of trouble sleeping among women of childbearing age in the United States. *Matern Child Health J*. 2017;21:306-314.
13. Chung J, Goodman M, Huang T, et al. Racial-ethnic differences in actigraphy, questionnaire, and polysomnography indicators of healthy sleep: the multi-ethnic study of atherosclerosis. *Am J Epidemiol*. 2021;193(1):107-120.
14. Makarem N, Shechter A, Carnethon MR, Mullington JM, Hall MH, Abdalla M. Sleep duration and blood pressure: recent advances and future directions. *Curr Hypertens Rep*. 2019;21:33.
15. Makarem N, Alcantara C, Williams N, Bello NA, Abdalla M. Effect of sleep disturbances on blood pressure. *Hypertension*. 2021;77:1036-1046.
16. Xu Y, Barnes VA, Harris RA, et al. Sleep variability, sleep irregularity, and nighttime blood pressure dipping. *Hypertension*. 2023;80(12):2621-2626.
17. Ross AJ, Yang H, Larson RA, Carter JR. Sleep efficiency and nocturnal hemodynamic dipping in young, normotensive adults. *Am J Physiol Regul Integr Comp Physiol*. 2014;307:R888-R892.
18. Bowman MA, Buysse DJ, Foust JE, Oyefusi V, Hall MH. Disturbed sleep as a mechanism of race differences in nocturnal blood pressure nondipping. *Curr Hypertens Rep*. 2019;21:51.
19. Covassin N, Bukartyk J, Singh P, Calvin AD, St Louis EK, Somers VK. Effects of experimental sleep restriction on ambulatory and sleep blood pressure in healthy young adults: a randomized crossover study. *Hypertension*. 2021;78:859-870.
20. Hoopes EK, Patterson F, Berube FR, et al. Actigraphy-derived rest–activity rhythms are associated with nocturnal blood pressure in young women. *J Hypertens*. 2021;39:2413-2421.
21. Bastien CH, Vallieres A, Morin CM. Validation of the insomnia severity index as an outcome measure for insomnia research. *Sleep Med*. 2001;2:297-307.
22. Cattano D, Sridhar S, Cai C, et al. Assessing risk of obstructive sleep apnea by STOP-BANG questionnaire in an adult surgical population screened in the preoperative anesthesia clinic. *Minerva Anestesiol*. 2016;82:605-606.
23. Hoopes EK, Brewer B, Robson SM, et al. Temporal associations between nightly sleep with daytime eating and activity levels in free-living young adults. *Sleep*. 2023;46(11):zsad123.
24. Carney CE, Buysse DJ, Ancoli-Israel S, et al. The consensus sleep diary: standardizing prospective sleep self-monitoring. *Sleep*. 2012;35:287-302.
25. Aili K, Astrom-Paulsson S, Stoetzer U, Svartengren M, Hillert L. Reliability of actigraphy and subjective sleep measurements in adults: the design of sleep assessments. *J Clin Sleep Med*. 2017;13:39-47.
26. Ancoli-Israel S, Martin JL, Blackwell T, et al. The SBSM guide to actigraphy monitoring: clinical and research applications. *Behav Sleep Med*. 2015;13(1):S4-S38. Suppl.
27. Patel SR, Weng J, Rueschman M, et al. Reproducibility of a standardized actigraphy scoring algorithm for sleep in a US Hispanic/Latino Population. *Sleep*. 2015;38:1497-1503.
28. Muntner P, Shimbo D, Carey RM, et al. Measurement of blood pressure in humans: a scientific statement from the American heart association. *Hypertension*. 2019;73:e35-e66.
29. Turner JR, Viera AJ, Shimbo D. Ambulatory blood pressure monitoring in clinical practice: a review. *Am J Med*. 2015;128:14-20.
30. Wolff-Hughes DL, Fitzhugh EC, Bassett DR, Churilla JR. Total activity counts and bouted minutes of moderate-to-vigorous physical activity: relationships with cardiometabolic biomarkers using 2003–2006 NHANES. *J Phys Act Health*. 2015;12:694-700.
31. Colley R, Connor Gorber S, Tremblay MS. Quality control and data reduction procedures for accelerometry-derived measures of physical activity. *Health Rep*. 2010;21:63-69.
32. Kenney MJ, Seals DR. Postexercise hypotension. Key features, mechanisms, and clinical significance. *Hypertension*. 1993;22:653-664.
33. Allen NB, Siddique J, Wilkins JT, et al. Blood pressure trajectories in early adulthood and subclinical atherosclerosis in middle age. *JAMA*. 2014;311:490-497.
34. Papathanasiou G, Zerva E, Zacharis I, et al. Association of high blood pressure with body mass index, smoking and physical activity in healthy young adults. *Open Cardiovasc Med J*. 2015;9:5-17.
35. Lascaux-Lefebvre V, Ruidavets J, Arveiler D, et al. Influence of parental history of hypertension on blood pressure. *J Hum Hypertens*. 1999;13:631-636.
36. Hartley TR, Sung BH, Pincomb GA, Whitsett TL, Wilson MF, Lovallo WR. Hypertension risk status and effect of caffeine on blood pressure. *Hypertension*. 2000;36:137-141.
37. Sayk F, Becker C, Teckentrup C, et al. To dip or not to dip: on the physiology of blood pressure decrease during nocturnal sleep in healthy humans. *Hypertension*. 2007;49:1070-1076.
38. Pengo MF, Won CH, Bourjeily G. Sleep in women across the life span. *Chest*. 2018;154:196-206.
39. Clark I, Landolt HP. Coffee, caffeine, and sleep: a systematic review of epidemiological studies and randomized controlled trials. *Sleep Med Rev*. 2017;31:70-78.
40. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *J Pers Soc Psychol*. 1986;51:1173-1182.
41. Matthews KA, Kamarck TW, HH M, et al. Blood pressure dipping and sleep disturbance in African-American and Caucasian men and women. *Am J Hypertens*. 2008;21:826-831.
42. Sherwood A, Routledge FS, Wohlgemuth WK, Hinderliter AL, Kuhn CM, Blumenthal JA. Blood pressure dipping: ethnicity, sleep quality, and sympathetic nervous system activity. *Am J Hypertens*. 2011;24:982-988.
43. D'Agata MN, Hoopes EK, Berube FR, et al. Evidence of reduced peripheral microvascular function in young black women across the menstrual cycle. *J Appl Physiol*. 2021;131(6):1783-1791. 1985. Under Review.
44. D'Agata MN, Hoopes EK, Berube FR, Hirt AE, Witman MA. Young black women demonstrate impaired microvascular but preserved macrovascular function compared to white women. *Exp Physiol*. 2021;106:2031-2037.
45. Hoopes EK, Berube FR, D'Agata MN, et al. Sleep duration regularity, but not sleep duration, is associated with microvascular function in college students. *Sleep*. 2021;44(2):zsaa175.
46. Hines AL, Thorpe RJ. Abstract 002: racial disparities in hypertension among young, black and white women, 1999–2014 national health and nutrition examination surveys. *Hypertension*. 2019;74:A002-A002.
47. Wang Y, Mei H, Jiang YR, et al. Relationship between duration of sleep and hypertension in adults: a meta-analysis. *J Clin Sleep Med*. 2015;11:1047-1056.

48. Marino M, Li Y, Rueschman MN, et al. Measuring sleep: accuracy, sensitivity, and specificity of wrist actigraphy compared to polysomnography. *Sleep*. 2013;36:1747-1755.
49. Booth JN, Muntner P, Abdalla M, et al. Differences in night-time and daytime ambulatory blood pressure when diurnal periods are defined by self-report, fixed-times, and actigraphy: improving the Detection of Hypertension study. *J Hypertens*. 2016;34(2):235-243.
50. Monte M, Cambao M, Mesquita Bastos J, Polonia J. Reproducibility of ambulatory blood pressure values and circadian blood pressure patterns in untreated subjects in a 1–11 month interval. *Rev Port Cardiol*. 2015;34:643-650.

How to cite this article: D'agata MN, Hoopes EK, Keiser T, et al. Device-estimated sleep metrics do not mediate the relation between race and blood pressure dipping in young black and white women. *J Clin Hypertens*. 2024;1-11. https://doi.org/10.1111/jch.14856