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Disturbed Sleep as a Mechanism of Race Differences in Nocturnal Blood Pressure Non-Dipping

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Abstract

Purpose of Review Disturbed sleep may be a mechanism of race differences in nocturnal blood pressure non-dipping. In support of this proposal, we summarize recent research from three literatures: (1) race differences (Black compared with White individuals) in nocturnal blood pressure non-dipping, (2) the association between disturbed sleep and nocturnal blood pressure non-dipping, and (3) race differences in disturbed sleep.

Recent Findings Black individuals are nearly twice as likely to have blood pressure non-dipping profiles compared with White individuals. This may be explained, in part, by sleep; shorter sleep duration, greater sleep fragmentation, less slow-wave sleep, and obstructive sleep apnea have each been associated with nocturnal blood pressure non-dipping. These sleep disturbances, in turn, are more common in Black compared with White individuals. Studies focused on nocturnal blood pressure non-dipping rarely assess sleep, and experimental evidence linking disturbed sleep with nocturnal blood pressure non-dipping in Black individuals is lacking.

Summary While mounting evidence from independent literatures suggests that disturbed sleep is a plausible, modifiable mechanism of race differences in nocturnal blood pressure non-dipping, definitive conclusions are premature given the current state of science.

Keywords Race · Sleep duration · Sleep apnea · Slow-wave sleep · Sleep efficiency · Blood pressure dipping

Introduction

African American/Black (hereafter referred to as Black) individuals are at increased risk for early mortality compared with non-Hispanic Caucasian American/White (hereafter referred to as White) individuals [1]. Cardiovascular disease-related mortality is one of the strongest predictors of this health disparity [2]. Race differences in the prevalence of cardiovascular disease risk factors may, in turn, contribute to race differences in the pathophysiology and clinical course of cardiovascular disease. Epidemiological studies of traditional cardiovascular disease risk factors have shown, for example, increased prevalence of physical inactivity [3], obesity [4], diabetes [5], and hypertension [3] in Black individuals compared with White individuals. Blunted nocturnal blood pressure dipping is a more newly recognized risk factor for cardiovascular disease. Defined as a decrease in nighttime blood pressure of less than 10% relative to daytime blood pressure, a meta-analysis of ten studies with 17,312 participants indicated that individuals who had blood pressure non-dipping were at increased risk for stroke, cardiovascular disease-related mortality, and all-cause mortality [6]. Another meta-analysis of 13 studies with 2753 participants showed that nocturnal blood pressure non-dipping was associated with higher common carotid intima media thickness, a proximal measure of atherosclerosis, compared with nocturnal blood pressure dipping [7]. Mounting evidence suggests that nocturnal blood pressure non-dipping may be a better predictor

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of cardiovascular disease risk than daytime blood pressure [8]. Important to the present review, nocturnal blood pressure non-dipping is nearly twice as common among Black compared with White individuals [9, 10].

Identification of modifiable pathways that contribute to racial disparities in cardiovascular disease is critical to reducing excess cardiovascular morbidity and mortality in Black men and women. Sleep, which is a multidimensional biobehavioral state [11], may represent one such modifiable pathway, as illustrated in Fig. 1. This review summarizes three disparate literatures to evaluate the hypothesis that disturbed sleep is an important biobehavioral pathway linking race to nocturnal blood pressure non-dipping. In keeping with the aims of *Current Hypertension Reports* to provide updates on recent publications, evidence reviewed here is primarily drawn from studies published between 2016 and 2019. First, we summarize evidence that blood pressure non-dipping is more common in Black compared with White individuals. We then summarize research suggesting that four key sleep disturbances are associated with nocturnal blood pressure non-dipping. Finally, we present evidence that these sleep disturbances are more prevalent in Black compared with White individuals. Although the current state of science cannot address questions of causality, we also summarize two recent cross-sectional studies that tested the extent to which sleep disturbances (statistically) account for race differences in nocturnal blood pressure non-dipping. Integration of these disparate literatures in this review may be used to guide definitive research on the extent to which disturbed sleep is an important, and modifiable, biobehavioral risk factor for racial differences in diurnal blood pressure profiles and their downstream effects on cardiovascular disease morbidity and mortality.

Race Differences in Nocturnal Blood Pressure Non-Dipping

A meta-analysis of 18 studies including 2852 participants showed that Black individuals from the USA had higher daytime and nighttime systolic and diastolic blood pressure, and experienced less of a nocturnal dip in both systolic and diastolic blood pressure compared with White individuals [12]. More recent studies have largely replicated these results [9, 10, 13, 14]. For example, studies that evaluated race differences in dipping status reported that nocturnal blood pressure non-dipping was more prevalent among Black (44–53%) compared with White individuals (27–34%) [9, 10, 15•]. Among studies that evaluated diurnal blood pressure profiles as a continuous variable, each reported a significant blunting of nocturnal blood pressure dipping in Black compared with White individuals [9, 10, 15•, 16]. In summary, converging evidence suggests that nocturnal blood pressure non-dipping is more common in Black compared with White individuals.

Though 24-h ABP monitoring is a powerful technique for measuring diurnal blood pressure patterns, findings from ABP studies may be affected by methodological choices, most notably how nighttime and daytime intervals are defined. For example, researchers/clinicians can specify the nocturnal period as a fixed interval (e.g., 12 am–6 am is suggested by the International Database of Ambulatory Blood Pressure [17••]) that is consistent across individuals, or tailor the nocturnal period to individuals' sleep time as identified by self-report or behavior (e.g., from wrist actigraphy). In a direct comparison of these methods, the fixed interval approach was found to generate minor systematic biases in non-dipping classification compared to tailored intervals defined by self-reported or actigraphy-assessed sleep intervals [17••]. There was greater

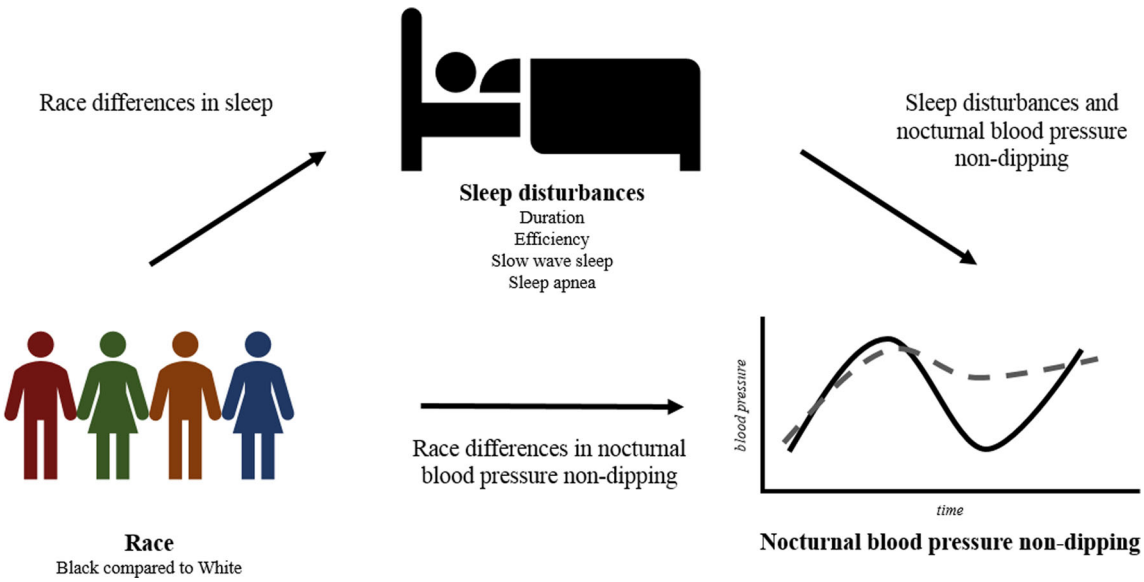

Fig. 1 A model of the reviewed literature suggesting that sleep disturbances are a mechanism of race differences in nocturnal blood pressure non-dipping

non-dipping classification agreement when comparing self-reported to actigraphy-assessed sleep intervals, suggesting that self-reported sleep intervals may be adequate for ABP studies. Although the use of fixed intervals has practical advantages such as reduced burden on patients/participants, sleep-defined intervals provide greater certainty that ABP measures occur during waking or sleep. Certainty regarding wakefulness and sleep may be especially important when evaluating race differences in diurnal blood pressure given known differences in sleep timing and duration in Black compared to White individuals [18]. The use of sleep-defined intervals provides clinicians and researchers the ability to disentangle the influence of sleep and endogenous circadian rhythms on race differences in diurnal blood pressure profiles.

A second methodological consideration in 24-h ABP monitoring is whether nighttime blood pressure assessments disturb sleep. Blood pressure is typically sampled more frequently during the day than it is at night [9, 10, 15•, 16], which seems to be due to concerns that the sphygmomanometer disturbs sleep. Empirical studies provide conflicting evidence regarding the effects of cuff inflation on diary- and actigraphy-assessed sleep, as well as possible race differences in sleep disruption related to cuff inflation [19, 20]. One possible reason for this conflicting evidence may be the age of the participants—college students may be more vulnerable to cuff-related sleep disruption [19] than middle-aged adults [20]. At present, it is unclear whether cuff inflation disrupts sleep and the extent to which cuff-related sleep disruption differs as a function of race.

Sleep Disturbances and Nocturnal Blood Pressure Non-Dipping

Endogenous circadian rhythms [21] and sleep work in concert to influence nocturnal physiology including parasympathetic nervous system dominance [22], diurnal nadirs in adrenocorticotropin (ACTH) and cortisol [23] levels, and rostral fluid shifts [24]. Disturbed sleep perturbs these physiological systems, with direct effects on blood pressure, including diurnal blood pressure profiles. For example, short sleep duration alters 24-h sympatho-vagal balance by decreasing parasympathetic activity and increasing sympathetic activity [22]. Experimental studies also show that acute sleep deprivation and restriction have marked effects on blood pressure. Significant increases in daytime and nighttime systolic blood pressure have been observed in response to total sleep deprivation [25–27] as well as one night of restricting sleep to 4 h [28]. Disturbances in the continuity and depth of sleep, as well as obstructive sleep apnea, may also influence diurnal blood pressure profiles.

Sleep continuity disturbances, characterized by difficulty initiating or maintaining sleep, are associated with physiological arousal such as decreased parasympathetic nervous system activity and increased cortisol levels [29]. Observational studies have reported blunted systolic blood pressure dipping in association with actigraphy-assessed decrements in sleep efficiency [30], and increased sleep fragmentation [31]. Similarly, laboratory sleep studies have shown that a greater number of arousals from sleep [31] and more wakefulness after sleep onset [32] were associated with blunted systolic and diastolic blood pressure dipping, respectively. Experimental data have also linked sleep fragmentation with nocturnal blood pressure, showing that noise-induced sleep fragmentation was associated with blunted systolic blood pressure dipping [33]. Thus, convergent observational, laboratory, and experimental evidences demonstrate that sleep continuity disturbances are associated with blunted nocturnal blood pressure dipping.

Polysomnographic sleep studies suggest that sleep architecture is linked to nocturnal blood pressure. Slow-wave sleep, also referred to as deep sleep, is the stage of sleep most clearly characterized by parasympathetic nervous system predominance [34]. Slow-wave sleep is also linked to nocturnal nadirs in blood pressure [34]. Moreover, decreased slow-wave sleep has been associated with increased nocturnal mean arterial pressure in both observational [32], cf. [31] and experimental studies [35]. Although the expense and participant burden associated with polysomnography have limited examination of the extent to which sleep architecture influences diurnal blood pressure profiles, data suggest that slow-wave sleep is an important driver of decreased nocturnal blood pressure.

In contrast to the paucity of studies examining sleep architecture and diurnal blood pressure profiles, numerous studies have evaluated the influence of obstructive sleep apnea on blood pressure dipping. Intermittent hypoxia [36], shifts in blood volume [24], and sympathetic nervous system activation [37] may contribute to increased blood pressure in patients with sleep apnea, including elevated daytime blood pressure [38] and greater prevalence of nocturnal blood pressure non-dipping [39]. One prospective study reported a dose–response relationship between severity of obstructive sleep apnea and risk for incident nocturnal blood pressure non-dipping at 7-year follow-up [40]. Moreover, treatment of sleep apnea with continuous positive airway pressure (CPAP) has been associated with clinical improvements in blood pressure. A meta-analysis of 44 studies with 4289 patients reported a reduction in daytime systolic blood pressure for patients in the CPAP condition relative to an inactive control [41], and an experimental study demonstrated that CPAP lowered nocturnal blood pressure in patients who previously were non-dippers [42•]. This large body of evidence strongly suggests that obstructive sleep apnea adversely affects diurnal blood pressure profiles.

Several critical questions remain regarding associations between disturbed sleep and diurnal blood pressure profiles. First, are sleep disturbances other than sleep apnea [40]

prospectively linked to the development of nocturnal blood pressure non-dipping? What are the independent and synergistic effects of disturbed sleep on diurnal blood pressure profiles? Previous observational and experimental evidences have examined these indices of sleep independently, but combinations of these measures (as conceptualized in the sleep health model [11]) may be even more important to diurnal blood pressure profiles. Finally, with the goal of identifying modifiable pathways that influence blood pressure non-dipping, do interventions that ameliorate short, fragmented, or light sleep result in blood pressure improvements, including increased nocturnal blood pressure dipping?

Race Differences in Sleep

Studies have consistently reported marked race differences in sleep [18] including shorter sleep duration, greater sleep fragmentation, alterations in sleep architecture, and greater prevalence of sleep apnea in Black individuals compared with White individuals. Race differences in disturbed sleep have been observed in men and women and across the lifespan, including children, adolescents, and adults.

Numerous studies have examined race differences in sleep duration. Recent epidemiological studies of adults in the USA found that Black individuals were more likely to report short sleep duration compared with White individuals [43, 44]. Data based on actigraphy-assessed sleep duration are more equivocal. While two recent studies of adults reported shorter total sleep time in Black compared with White individuals [45•], [46], two other studies found no significant differences in actigraphy-assessed sleep duration [47, 48]. A meta-analysis of eight studies including 1523 participants found that Black individuals had shorter objectively assessed sleep duration compared with White individuals [18]. Similar to studies in adults, a recent review of race differences in children's sleep noted that 17 of 18 studies found that Black youth have shorter sleep duration compared with White youth [49••]. In sum, a large literature demonstrates that Black individuals have shorter sleep duration compared with White individuals across the lifespan.

Poorer sleep continuity has also been observed in Black compared with White individuals. Different components of sleep continuity, including greater actigraphy-assessed wake after sleep onset [45•, 48] lower sleep efficiency [48], and greater sleep fragmentation [48] have been observed in Black compared with White individuals. Data in children are less consistent, with some studies reporting greater actigraphy-assessed sleep continuity disturbances in Black compared with White adolescents [50], and others reporting no significant differences among Black and White children [51]. Taken as a whole, these data suggest that sleep continuity disturbances are common in Black adults, with more equivocal findings in children.

Polysomnographic sleep studies also show marked race differences in sleep architecture. A meta-analysis of eight studies in 980 adults reported lower levels of slow-wave sleep in Black compared with White individuals [18]. Studies in adult samples have consistently reported that Black adults exhibit approximately 50% less slow-wave sleep compared to age- and sex-equated White adults [52–54]. To our knowledge, only one study has examined race differences in slow-wave sleep in adolescents. This study reported no significant difference in slow-wave sleep for Black compared with White individuals, though it is worth noting that the study only included 15 Black participants and, thus, may have been underpowered to examine race differences in sleep [55]. Studies in adolescents and children are critical for evaluating race differences in slow-wave sleep across the lifespan.

Obstructive sleep apnea is more prevalent in Black compared with White individuals. In a meta-analysis, Black individuals were more likely to have sleep-disordered breathing (five studies and over two million participants), as well as a higher apnea-hypopnea index (eight studies and over 6000 participants), compared with White individuals [56]. Adherence to CPAP for obstructive sleep apnea is lower in Black compared with White individuals [57], suggesting that alternate strategies may be warranted to improve treatment of apnea and/or compliance with CPAP in Black individuals. Although many of the studies included in the meta-analysis [56] adjusted for factors that might account for race differences in obstructive sleep apnea, more careful examination is needed to evaluate factors that underlie race differences in disturbed sleep including shorter sleep duration, greater sleep fragmentation, and lower levels of slow-wave sleep.

In summary, race differences in sleep are marked and consistent across studies. The effects of physiological, psychological, social, cultural, and environmental factors that underlie race differences in sleep are less well understood, though some studies are beginning to probe these factors (e.g., [58•]). It is also worth considering whether these sleep differences merit clinical intervention. Obstructive sleep apnea and short sleep duration are compelling intervention targets, given meta-analytic evidence that these measures increase risk for cardiovascular disease morbidity and mortality [59, 60]. However, it is less clear whether interventions to increase slow-wave sleep, or increase sleep continuity given that Black individuals are less likely to report symptoms of insomnia [56], are warranted. Identifying the myriad pathways that contribute to disturbed sleep in Black individuals can inform primary prevention efforts, triage health services, and tailor interventions to improve sleep and reduce race differences in disturbed sleep.

Race, Sleep, and Blood Pressure Non-Dipping

We posit that disturbed sleep is an important, modifiable pathway through which race influences diurnal blood pressure profiles, including increased nocturnal blood pressure non-dipping in Black compared with White individuals (see Fig. 1). This model is based on three separate literatures: race differences in nocturnal blood pressure non-dipping; sleep

disturbances and nocturnal blood pressure non-dipping; and race differences in sleep. To our knowledge, only two studies have evaluated all three components of this model, yet both were limited by their cross-sectional design. A study of 55 Black and 66 White adults reported that a composite measure of sleep quality statistically attenuated the association between race and nocturnal blood pressure non-dipping [61]. In contrast, a study by Hughes and colleagues reported that sleep duration and fragmentation did not explain observed race differences in diurnal blood pressure profiles [19]. Prospective and experimental studies are needed to directly test the hypothesis that disturbed sleep contributes causally to race differences in nocturnal blood pressure non-dipping.

Conclusion

Black individuals are at higher risk for cardiovascular disease morbidity and mortality compared with White individuals. One reason for this difference may be nocturnal blood pressure non-dipping, a risk factor for cardiovascular disease. The prevalence of nocturnal blood pressure non-dipping is nearly double in Black compared with White individuals. Identifying modifiable pathways linking race and nocturnal blood pressure non-dipping is critical for ameliorating differences in cardiovascular disease. As shown in the conceptual model that underlies this review, we propose that disturbed sleep is a key, modifiable pathway through which Black individuals are at increased risk for nocturnal blood pressure non-dipping, compared with White individuals. Mounting evidence suggests that disturbed sleep including short sleep duration, sleep fragmentation, decreased slow-wave sleep, and obstructive sleep apnea is associated with alterations in diurnal blood pressure profiles. These same sleep disturbances are more prevalent in Black compared with White individuals, including men and women across the lifespan. Longitudinal and experimental studies are needed to test this model directly. Interventions that ameliorate known race differences in sleep may potentially improve diurnal blood pressure profiles and, if effective, reduce excess cardiovascular morbidity and mortality in Black men and women.

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Compliance with Ethical Standards

Conflict of Interest Dr. Buysse reports personal fees from BeHealth, Emmi Solutions, American Academy of Physician Assistants, Bayer, CME Institute, and Ebb Therapeutics, outside the submitted work. In addition, Dr. Buysse received licensing fees for several research instruments whose copyright is held by the University of Pittsburgh and Ryerson University. The other authors declare no conflicts of interest relevant to this manuscript.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- •• Of major importance

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