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Disparities in Hypertension among African-Americans - Implications of Insufficient Sleep

Naima Covassin, Ph.D.¹, Eddie L. Greene, M.D.², Prachi Singh, Ph.D.¹, Virend K. Somers, M.D, Ph.D.¹

¹Department of Cardiovascular Medicine, Mayo Clinic, Rochester, MN

²Division of Nephrology and Hypertension, Department of Internal Medicine, Mayo Clinic, Rochester, MN

Abstract

Purpose of review: Sleep deficiency has been proposed as a potential contributor to racial disparities in cardiovascular health. We present contemporary evidence on the unequal burden of insufficient sleep in Blacks/African-Americans and the repercussions for disparate risk of hypertension.

Recent findings: The prevalence of insufficient sleep is high and rising, and has been recognized as an important cardiovascular risk factor. Presumably due to a constellation of environmental, psychosocial and individual determinants, these risks appear exacerbated in Blacks/African-Americans, who are more likely to experience short sleep than other ethnic/racial groups. Population-based data suggest that the risk of hypertension associated with sleep deficiency is greater in those of African ancestry. However, there is a paucity of experimental evidence linking short sleep duration to blood pressure levels in African-Americans.

Summary: Blacks/African-Americans may be more vulnerable to sleep deficiency and to its hypertensive effects. Future research is needed to unequivocally establish causality and determine mechanism underlying the postulated racial inequalities in sleep adequacy and consequent cardiovascular risk.

Keywords

African-Americans; nocturnal dipping; health disparities; lifestyle; sleep

Introduction

The encouraging downward trend in cardiovascular mortality observed since 2000 has now reversed, with recent statistics detecting an alarming uptick in age-adjusted deaths attributable to heart disease [1]. Stratification of the rise in cardiovascular fatalities during 2011–2014, according to race and ethnicity, revealed striking disparities, with a four-fold increment in the Black/African descendant population compared to Whites. These estimates

reaffirm the burden of cardiovascular disease disproportionately afflicting African-Americans. Cardiovascular mortality in Blacks indeed remains the highest among all racial/ethnic groups [2, 3] and accounts for more than 30% of the gap in life expectancy in Blacks when compared to Whites [4].

When considering the determinants of racially discrepant cardiovascular death rates, high blood pressure (BP) stands as the single most important culprit [5, 6]. Hypertension is highly prevalent in Blacks/African-Americans, affecting 42–44% of individuals of African descent vs 28–29% of Whites [7, 8], presents at a younger age [9], and is less frequently adequately controlled despite more aggressive treatment regimens [10]. Because hypertension-related complications are substantially greater in Blacks/African-Americans [3, 11], improving both prevention and management of high BP in this population is critical in reducing the potential morbidity and associated mortality. Among the variety of risk factors that are thought to favor these enduring disparities, and which may be preemptively targeted, growing evidence implicates sleep duration.

Chronic sleep deficiency has become a burgeoning phenomenon worldwide. The expansion of artificial illumination, occupational schedules, and around the clock service sectors, along with the advent and vast spread of electronic devices, and primarily mobile communication technology, is having an unprecedented impact on sleep habits. The time allocated to sleep has been progressively shortened in the past few decades, resulting in shorter sleep duration and greater prevalence of unmet sleep needs. Since 1985, the proportion of adults sleeping 6 or less hours has increased by 31% [12], and currently approaches 35% of the US population [13]. Departures from healthy sleep duration, defined by expert consensus panels as 7 to 9 hours of sleep [14, 15], have important repercussions for cardiovascular health, and related comorbidities, as documented by accumulating studies on the adverse effects of short sleep [16, 17].

These sleep duration trajectories and the consequences of sleep deficiency on health are markedly influenced by ethnic/racial status, and available data converge to indicate that they are more pronounced in African-Americans, thus presumably contributing to the unequal risk of hypertension and cardiovascular disease observed in this population.

Here we will review recent literature on the epidemiology of habitual short sleep in those of African ancestry, and highlight those aspects that may be involved in sleep-related racial disparities. Observational and experimental evidence supporting an unequal impact of insufficient sleep on hypertension risk in Blacks/African Americans will be discussed, along with underlying candidate mechanisms.

Epidemiology of sleep deficiency in Blacks/African-Americans

The ongoing sleep deprivation epidemic seen in the general population appears to disproportionately affect ethnic/racial minorities, especially in Blacks/African-Americans.

By examining data on sleep duration from 444,306 adults participating in the 2014 Behavioral Risk Factor Surveillance System (BRFSS), the Centers of Disease Control and Prevention [13] showed that the highest age-adjusted prevalence of healthy sleep duration

(defined as ≥ 7 hours of sleep in a 24-hour period) was observed among non-Hispanic Whites (66.8%), while only 54.2% of non-Hispanic Blacks reported sleeping at least 7 hours. Self-reported short sleep (<7 hours) was found in 38% of Black respondents to the 2004–2011 National Health Interview Survey (NHIS), compared to 28% of Whites [18]. The disparate prevalence of short sleep has been subsequently reproduced, examining different waves of the same nationwide survey [19, 20], and observed in other racially-diverse samples [21–23], which collectively show that Blacks sleep, on average, approximately 30 min less than Whites. Ethnic/racial disparities in sleep quantity are accentuated when curtailed sleep duration is modeled as short or very short sleep. Using the 2007–2008 National Health and Nutrition Examination Survey (NHANES), Whinnery et al [24] found that in Blacks/African-Americans relative to Whites, odds for short sleep (5–6 hours) and for very short sleep (<5 hours) were 1.85 and 2.34, respectively. Trend analysis using a similar classification expanded these results, showing that both very short and short sleep were consistently more frequent in Blacks across the years from 1977 through 2009 [20]. In line with prevalence data, longitudinal observations indicate that the likelihood of experiencing short sleep in the future is also more elevated in those of African ancestry. Compared to their White counterparts, African Americans exhibit a greater increment in the probability of short sleep (<6 hours) over time, which doubled over a 34 years follow-up [25]. This racial gap emerges at young ages [26, 27], and, while evident in both sexes and across age strata, it seems particularly stronger in young and middle-age Black men, who sleep 75–82 min less than White women [28, 29].

It is pertinent that the large majority of these population-based studies relied on self-reported information on sleep duration, which may be biased by the individuals' tendency to overestimate sleep, and only moderately correlates with sleep quantity as determined from actigraphy or polysomnography [30]. Nevertheless, divergent ethnic-dependent sleep durations are apparent, and possibly amplified, also when considering objective sleep measures [28, 31–35]. In the Multi-Ethnic Study of Atherosclerosis (MESA), actigraphy-derived habitual sleep duration in Blacks was the lowest amid all racial/ethnic groups, with 43.4% of them sleeping less than 6 hours (vs 19.3% of White) [28].

Similar to other health disparities, an array of environmental, psychosocial and behavioral factors has been proposed to account for the heightened probability of sleep deficiency exhibited in cohorts of African descent. Socioeconomic status is conventionally regarded as a major determinant of ethnic/racial-dependent disparities, and it has accordingly been invoked as a modulator of sleep deprivation risk. Neighborhood characteristics and household conditions may hinder adequate sleep because of environmental disturbances such as inappropriate illumination, crowding, excessive noise, air pollution, and uncomfortable sleeping arrangements [36–38]. Low safety and social fragmentation experienced in disadvantaged neighborhoods have been linked to poor health and insufficient sleep in multi-ethnic samples [31, 39, 40]. Other socioeconomic predictors of enhanced probability of abnormal sleep in Blacks/African-Americans likely include employment status, job strain, income, and educational attainment [24, 25, 41, 42]. Cultural beliefs also may influence sleep health practices and determine the quantity of sleep sought [43]. On the contrary, perceived racial discrimination, another leading health risk factor, has

been less consistently related to aberrant sleep duration patterns in African-Americans [32, 44].

Chronic stress, notably a common denominator of many of these risk factors, confers greater susceptibility to sleep deficiency [45] and may underlie the racial inequality in sleep duration. By evaluating data from 4,864 Black participants in the Jackson Heart Study, Johnson et al [46] found that lower sleep time and greater probability of short sleep were associated with elevated subjective stress burden.

Blacks/African-Americans are also more likely to engage in hazardous behaviors associated with insufficient sleep [47–49], such as smoking, excessive alcohol consumption, physical inactivity, and overeating [3].

Poor psychological and physical health may compromise adequate sleep and contribute to the observed inequalities. Chronic sleep debt may especially be secondary to comorbid sleep disorders, such as sleep disordered breathing and insomnia, which are demonstrated to be more prevalent in Blacks/African-Americans than in Whites [25, 28, 50–52]. Despite being likely implicated in the sleep duration disparities, environmental, social and individual attributes do not fully explain them, as factoring these variables in the analysis attenuates, but does not dissolve, the strength of the observed associations in most of the studies. Further support for the concept that these aspects are not the sole determinants of the exaggerated risk of short sleep experienced by those of African heritage arises from a study that used propensity score modeling [22]. In a biracial sample of 1,022 Black individuals from the UK Biobank, closely propensity score-matched to White subjects across a multitude of known covariates of sleep duration, Blacks exhibited more than three-fold greater odds of short sleep relative to Whites, echoing the findings from unmatched samples presented above.

Bearing in mind that habitual sleep duration is a heritable trait, and that a series of genetic variants have been associated with sleep quantity [53–56], it is plausible that genetic components may also play a role. The CHARGE Consortium Genome-Wide Association Study detected significant associations with sleep duration at two loci near IER3 and PAX8 in a large sample of individuals from European heritage, and replicated these findings in an African-American population [55]. Conversely, polymorphisms in the dopamine receptor D2 (DRD2) have been associated with sleep duration in an ethnically-diverse cohort with the exclusion of groups of African descent [54], in whom a non-significant yet opposite direction was observed instead. Though provocative, more evidence is needed to implicate genetic vulnerabilities in these sleep disparities.

Insufficient sleep and disparate risk of hypertension in Blacks/African-Americans

Observational data

The concept of sleep deficiency as a novel contributor to disparities in cardiovascular health has recently gained greater attention and an accompanying increase in supporting data in the literature. It is now recognized that insufficient sleep is an important independent risk factor

for morbidity and mortality. As summarized by recent meta-analyses, short sleep duration is associated with a host of cardiometabolic risk factors such as obesity [57] and type 2 diabetes [58], and predicts cardiovascular events and death [59, 60]. The linkage between insufficient sleep and hypertension is especially robust, with several population-based studies reporting a much higher percentage of hypertensives among short sleepers compared to normal sleepers [61–63]. Likewise, those who sleep ≤ 6 hours/night are more likely to develop future hypertension than those who sleep 7–8 hours [64–67]. By pooling adjusted data from 21 studies, Guo et al [66] found that short sleepers had 21% greater probability of prevalent hypertension relative to normal sleep, and similarly higher relative risk estimates for incident hypertension (RR= 1.23, 95% CI=1.06–1.42). Demographic aspects appear to moderate the probability of high BP associated with short sleep, as this link is more pronounced among young and middle-aged adults [62, 63] and in ethnic/racial minorities [34, 65, 68]. In regard to the latter group, Blacks/African Americans may be especially vulnerable to the heightened likelihood of hypertension evoked by insufficient sleep.

A cross-sectional examination of the 2009 NHIS, including 25,352 adults aged 18–85, revealed a significant interaction between race/ethnicity and sleep quantity with respect to hypertension risk [68]. In unadjusted analysis, odds for high BP were 34% greater in Blacks sleeping <6 hours compared to White short sleepers. This relationship retained significance after correcting for known sociodemographic, lifestyle and medical determinants of high BP, thus suggesting independence for each of the observed associations. Resistant hypertension in Blacks is more common among short sleepers than in those who sleep at least 7 hours [69]. A longitudinal study of the young and middle-aged participants of the Coronary Artery Risk Development in Young Adults (CARDIA) cohort showed that habitual sleep, as measured from actigraphy, mediated the greater BP elevation over time experienced by Blacks compared to Whites. Larger mediation effects were detected for diastolic BP (DBP) and in male participants, as controlling for sleep duration explained 84% of the excess change in DBP exhibited by Black men compared to White women. While these data suggest potentiated effects of short sleep in males, Curtis et al [34] found that sleep duration mitigated the race-dependent higher systolic (SBP) and DBP in Black women, while no differences were found among men. When BP was incorporated in a composite index comprising established cardiometabolic biomarkers such as waist circumference, insulin resistance, and C-reactive protein (CRP), in adjusted analysis reductions in the quantity of sleep explained a staggering 41% of the racial differential, with a particularly stronger association found in Black females. However, as acknowledged by the authors, these results may have been less robust because of the paucity of African-American male participants ($n=36$) and consequent statistical power limitations. Nevertheless, this study remains especially relevant as it underscores the differential impact of short sleep on numerous cardiometabolic health indicators, reinforcing prior findings.

In a stratified analysis of the 2007–2008 NHANES cohort by race/ethnicity, Grandner et al [70] found that the link between short sleep duration and circulating CRP was stronger in Blacks/African-Americans than in other ethnic /racial groups, and especially in men. Relative to normal sleep (7–8 hours), Blacks sleeping ≤ 5 hours had 76% increased probability of being overweight and 81% probability of being obese, while in Whites the same sleep duration was associated with 10% and 51% heightened risk, respectively [71].

Comparable, disparate odds for obesity in Black short sleepers (retained after multivariate adjustment) were reported by Donat et al [72]. Similar patterns for racial distribution have been observed when considering the relation between sleep duration and hypercholesterolemia [73] and diabetes [18]. These highly prevalent comorbid conditions are disproportionately present in those of African descent [3, 5], and may favor BP elevation offering a potentially plausible set of contributing factors to the causal chain between aberrant sleep and risk of hypertension.

Although the disproportionate probability of hypertension in conjunction with short sleep in Blacks/African-Americans withstands corrections for conventional sociodemographic, behavioral, and medical covariates, residual effects cannot be ruled out. Among the possible confounders that may be involved in the excessive risk of hypertension with short sleep, the presence of sleep disorders is perhaps most critical. Sleep disturbances may not only result in sleep deprivation but may also cause BP elevation and hypertension. In this respect, sleep apnea is a well-accepted determinant of hypertension in the general population and in African-Americans [3, 74]. However, taking into account sleep apnea indexes does not abolish blunted nocturnal BP decline (“dipping”) in African-Americans [75]. Conversely, controlling for poor sleep quality weakens the racial discrepancy in BP dipping [76], indicating that sleep difficulties may play a contributing role. The relevance of comorbid sleep disturbances to the relationship between sleep deficiency and hypertension in Blacks/African-Americans warrants further investigation.

Candidate Mechanisms

The predictive significance of sleep deficiency for hypertension risk as observed in population-based investigations is substantiated by laboratory studies of experimentally-induced sleep loss, yielding data suggesting causation. Acute elevations in BP are noted after 24–88 hours of total sleep deprivation [77–82], with increases in SBP shown more consistently [77, 81, 82]. Experimental models of prolonged partial sleep deprivation (also called sleep restriction), which approximate more closely the extent of sleep loss commonly experienced in real life, provide mixed results. Non-significant effects on resting SBP and DBP were recorded after 5 nights of <5 hours [83] or 4 hours of sleep [84, 85], while increases were found after 7 days of 3 hours of sleep/night [86]. Such conflicting findings could be in part ascribed to the various experimental conditions (e.g., magnitude and duration of sleep truncation, timing of BP measurement). An elegant study from Yang et al [87] demonstrated that recurrent exposure to 3-day bouts of sleep restriction (4 hours/night) distorts diurnal BP pattern by increasing daily average SBP and DBP and attenuating nocturnal dipping, with larger responses evident during early experimental sleep curtailment and more sustained for DBP.

Although the nature of the causal pathway linking sleep curtailment to BP elevation is yet to be fully elucidated, laboratory-based studies also provide insights into candidate mechanisms governing BP.

Insufficient sleep has been shown to perturb neural circulatory control, stimulating sympathetic activation and altering baroreflexes as mechanisms to both elicit and maintain BP elevation. Sympathoexcitation induced by sleep loss has been postulated primarily on the

basis of increased circulating norepinephrine [83, 88–90] observed following prolonged wakefulness, accompanied by increased sympathetic traffic to skeletal muscle [77–79], while heart rate changes remain conflicting [81, 83–86]. Baroreflex function has been shown to reset to a higher BP after sleep loss but sensitivity is largely retained [78, 79]. While the hemodynamic effects of sleep truncation on cardiac output and vascular resistance are unclear [77, 81, 84, 91], defective endothelium-dependent vasodilation in resistance and conductance vascular beds [83, 92, 93] are indicative of vascular dysfunction. Additionally, increased arterial stiffness as measured by pulse-wave velocity has been observed after 24-hours of sustained wakefulness [81]. Limited data on short-term total sleep deprivation are suggestive of aberrant renin-angiotensin-aldosterone system stimulation, manifested by suppression of nocturnal increases in aldosterone [94], renin, and angiotensin II [95]. Abnormal sodium and water handling is further indicated by exaggerated nocturnal diuresis and natriuresis seen under conditions of controlled sodium and water intake during acute [95] and prolonged sleep loss [87]. Furthermore, cardiovascular reactivity to physical and mental stressors, an established precursor of hypertension [96–98], is potentiated in sleep restricted subjects, as indicated by enhanced BP, heart rate and cortisol responses [99–101].

Whether these findings can be extrapolated to diverse populations and specifically to those of African ancestry is currently unknown, because the ethnic/racial composition of those samples was not reported, and, to our knowledge, racial-specific effects of experimentally induced sleep curtailment on BP have not been examined in current literature. Nevertheless, it is tempting to speculate that racial-dependent effects of experimental sleep truncation would emerge, validating epidemiological observations. A number of considerations give strength to this conjecture.

First, nighttime appears to be an especially vulnerable period in Blacks/African-Americans. Studies using ambulatory BP monitoring to delineate 24-hour BP profile and phenotypes have showed that racial gaps in high BP are amplified when considering nocturnal BP. Prevalence of nocturnal hypertension is more than double in those of African descent than in Whites [102]. BP decline during nighttime is often blunted in African-Americans, and non-dipping status is present in 51.5–44.9% of them vs 33–26.7% of Whites [102–104], irrespective of BP status. In normotensives, BP dipping predicts progression towards prehypertension and hypertension [104]. Given this time-dependent susceptibility, it is plausible that the alterations in 24-hour BP dynamics and reduced dipping induced by sleep loss may be exacerbated in Blacks/African-Americans, thus promoting, in the long term, development of sustained hypertension.

Second, as it pertains to BP regulatory pathways, many of the mechanisms found to be affected by sleep deprivation are known to be implicated in the racial disparities in hypertension, with data suggesting alterations in normotensive Blacks/African-Americans. Reflex and autonomic BP control is impaired in Blacks/African-Americans relative to Whites, as indicated by blunted carotid baroreflex responsiveness to hypertensive stimuli [105], upregulated alpha-adrenergic receptor sensitivity [106, 107] coupled with depressed beta-adrenergic receptor responsiveness [107, 108], enhanced sympathetic vascular transduction [109], and reduced nocturnal decline in epinephrine and norepinephrine excretion [76, 110]. Functional and structural vascular deterioration in Blacks/African-

Americans is further expressed as impaired endothelial responsiveness in the micro- and macro-circulation [111, 112], heightened systemic and minimum forearm vascular resistance [113], and greater arterial stiffness [112, 114]. Aberrant renin-angiotensin system and imbalanced electrolyte homeostasis, with elevated prevalence of salt sensitivity, are more common substrates of hypertension in African-Americans [115, 116]. Interestingly, potentiated reactivity to stressors is classically regarded as a main contributor for disparate risk of hypertension in Blacks/African-Americans. Relative to Whites, those of African descent exhibit augmented cardiovascular reactivity to physical and mental stress [117]. This exaggerated response predicts future BP elevation better than resting values [118], and is more predictive in Blacks than in Whites [119]. This intermediary mechanism may be especially relevant because insufficient sleep in itself can be regarded as a stressor [120]. As a result of increased allostatic load, the cardiovascular hyper-reactivity induced by sleep curtailment may be further amplified in those of African heritage, hence unequally predisposing Blacks/African-Americans to hypertension. Of note, abnormal hypothalamic-pituitary-adrenal axis (HPA) activation, a sentinel of maladaptive stress response and poor health [121], is associated with attenuated BP dipping responses in Black women [122] and can be partially attributed to racial differences in sleep duration in African-Americans in comparison to Whites [33].

Third, controlled laboratory studies have reported a broad spectrum of behavioral, physiological and molecular modifications in response to sleep deprivation, which may act in concert to contribute to altering cardiovascular activity, producing BP elevation. Sleep curtailment has been shown to impair whole-body and tissue insulin sensitivity [90, 123–125], increase leptin [125–127] and energy intake [126, 128, 129], stimulate HPA axis [101, 125, 130], and promote inflammation [82, 131]. Notably, very few studies have disclosed the ethnic/racial makeup and/or reported differential effects in African-Americans compared to Whites.

Obesity is an important precursor of hypertension in the general population and thought to be involved in the hypertension disparities. In a large in-laboratory study, the magnitude of weight gain recorded after five days of experimental sleep restriction (4 hours of sleep/night) was significantly greater in African-Americans than in Whites, with the largest difference (almost five-fold) being between African-American males and Caucasian females [129]. It is intriguing that this discrepant metabolic response to sleep restriction mirrors the prevalence data showing the largest gap in risk between these two racial and sex groups. Compared to Whites, the calorie intake in African Americans undergoing sleep restriction was similar, but they consumed more calories from carbohydrates, and specifically from sweetened beverages [132]. Excessive sugar intake may be partially responsible for the racial gap in diabetes and cardiovascular disease [133]. These experimental data also align with population survey data documenting that Blacks/African-Americans may be more likely to report unhealthy nutritional choices, and the discrepant BP levels compared to Whites can be partially attributed to dietary patterns [134]. African-Americans are especially sensitive to the effects of sodium and potassium intake on BP regulation [135]. In this respect, lower potassium consumption, often reported by Black/African-American subjects [136], has been associated with short sleep duration in the NHANES cohort [48].

As it pertains to neurohormonal underpinnings of BP increased, sympathetic activation induced by experimental sleep restriction may be mediated by leptin increases [137]. In a racially-diverse study, Simpson found comparable increases in plasma leptin levels following 5 nights of 4 hours of sleep in African-Americans and Caucasians [127]. More puzzling are the changes noted in adiponectin, an anti-inflammatory and anti-atherogenic adipocytokine thought to protect against hypertension [138]. While adiponectin levels decreased in White women following sleep curtailment, it increased in their Black counterparts [139]. Whether these data relate to different body fat distribution, or imply a compensatory protective response, is unclear. Regardless, they reaffirm the pressing need for factoring in ethnicity when investigating the impact of sleep loss on biological function.

Conclusions

Insufficient sleep has profound, pernicious effects on cardiovascular health and has been proposed to be implicated in cardiovascular health disparities. Epidemiological evidence is strongly suggestive of a widespread and disproportionate prevalence of short sleep among those of African heritage, presumably due to a constellation of unfavorable environmental, psychosocial, behavioral, and possibly biological/genetic traits. The heightened likelihood of sleep deficiency exhibited by Blacks/African-Americans has important public health implications, being associated with excessive predisposition to cardiometabolic derangements and especially development of hypertension. However, in spite of suggestive arguments, and in absence of corroborating experimental causative data, the linkage between hypertension and sleep deficiency in this racial group remains inferential. This critically important knowledge gap mandates powered research studies and representative cohort sampling of Blacks/African-Americans, to test the impact of experimentally-induced sleep loss on abnormal BP alternations in this racial group. Gender/age interactions are also important to evaluate, as well as the identification of biological mechanisms, so to determine postulated disparate BP responses to sleep loss in this population. In-laboratory controlled studies should be integrated with investigations in the field to comprehensively identify correlates of hypertension in association with short sleep among Blacks/African-Americans. A deeper understanding of this relationship may provide the basis for racially-sensitive, preemptive and therapeutic strategies to rectify poor sleep habits and combat chronic sleep debt, as strategies to mitigate the unequal burden of hypertension and cardiovascular disease in patients and subjects of African descent.

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