Current Hypertension Reports (2018) 20: 57
https://doi.org/10.1007/s11906-018-0855-1

SECONDARY HYPERTENSION: NERVOUS SYSTEM MECHANISMS (M WYSS, SECTION EDITOR)

Disparities in Hypertension Among African-Americans: Implications of Insufficient Sleep

Naima Covassin[1] · Eddie L. Greene[2] · Prachi Singh[1] · Virend K. Somers[1]

Published online: 8 June 2018

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 the 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 fourfold 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,

This article is part of the Topical Collection on *Secondary Hypertension: Nervous System Mechanisms*

✉ Naima Covassin
Covassin.naima@mayo.edu

[1] Department of Cardiovascular Medicine, Mayo Clinic College of Medicine, 200 First Street SW, Rochester, MN 55905, USA

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

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 h 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 hypertensions 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 disproportionally 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 h of sleep in a 24-h period) was observed among non-Hispanic Whites (66.8%), while only 54.2% of non-Hispanic Blacks reported sleeping at least 7 h. Self-reported short sleep (< 7 h) 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 h) and for very short sleep (< 5 h) 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 h) over time, which doubled over a 34-year 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, 31–33, 34•, 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 h (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 multiethnic 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 4864 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 1022 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 threefold 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 nonsignificant 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 h/night are more likely to develop future hypertension than those who sleep 7–8 h [64, 65, 66•, 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 h 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 h [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 h), Blacks sleeping ≤ 5 h 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 do 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 h 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. Nonsignificant effects on resting SBP and DBP were recorded after 5 nights of < 5 h [83] or 4 h of sleep [84, 85], while increases were found after 7 days of 3 h 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 h/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] is indicative of vascular dysfunction. Additionally, increased arterial stiffness as measured by pulse-wave velocity has been observed after 24 h 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-h 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 nondipping status is present in 51.5–44.9% of them vs 33–26.7% of Whites [102•, 103, 104], irrespective of BP status. In normotensives, BP dipping predicts progression toward prehypertension and hypertension [104]. Given this time-dependent susceptibility, it is plausible that the alterations in 24-h 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 macrocirculation [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 hyperreactivity 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 the 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 5 days of experimental sleep restriction (4 h of sleep/night) was significantly greater in African-Americans than in Whites, with the largest difference (almost fivefold) 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 et al. found comparable increases in plasma leptin levels following 5 nights of 4 h 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 the 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 as 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.

Funding Information Dr. Covassin is supported by NIH HL134808 and HL065176, American Heart Association grant 16SDG27250156, and Mayo Clinic Center for Clinical and Translational Science grant Marie Ingalls Cardiovascular Research Career Development Fund in honor of Dr. Alexander Schirger. Dr. Singh is supported by American Heart Association grant 17GRNT33660138 and NIH HL65176. Dr. Somers is supported by NIH HL134808 and HL065176. The contents of this article are solely the responsibility of the authors and do not necessarily represent the official view of the NIH.

Compliance with Ethical Standards

Conflict of Interest Dr. Somers has served as a consultant for GlaxoSmithKline, Dane Garvin, ResMed, Respicardia, Philips, Bayer, and U Health; has received grant support from a Philips Respironics Foundation gift to Mayo Foundation; and is working with Mayo Health Solutions and their industry partners on intellectual property related to sleep and cardiovascular disease. The other authors indicate no conflicts of interest.

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.

References

Papers of particular interest, published recently, have been highlighted as:
- Of importance

1. Heron M, Anderson RN. Changes in the leading cause of death: recent patterns in heart disease and cancer mortality. NCHS Data Brief. 2016;254:1–8.
2. Sidney S, Quesenberry CP Jr, Jaffe MG, Sorel M, Nguyen-Huynh MN, Kushi LH, et al. Recent trends in cardiovascular mortality in the United States and public health goals. JAMA Cardiol. 2016;1(5):594–9.
3.• Carnethon MR, Pu J, Howard G, Albert MA, Anderson CAM, Bertoni AG, et al. Cardiovascular health in African Americans: a scientific statement from the American Heart Association. Circulation. 2017;136(21):e393–423. **This statement provides an up-to-date summary of the disparate burden of cardiovascular diseases in African-Americans and emphasizes the use of racial-sensitive therapeutic approaches to cardiovascular disease management.**
4. Gillespie CD, Wigington C, Hong Y. Coronary heart disease and stroke deaths—United States, 2009. MMWR Surveill Summ. 2013;62(Suppl 3):157–60.
5. Benjamin EJ, Blaha MJ, Chiuve SE, Cushman M, Das SR, Deo R, et al. Heart disease and stroke statistics—2017 update: a report from the American Heart Association. Circulation. 2017;135(10):e146–603.
6. Kung HC, Xu J. Hypertension-related mortality in the United States, 2000–2013. NCHS Data Brief. 2015;193:1–8.
7. National Center for Health Statistics. Health, United States, 2016: With chartbook on long-term trends in health. Hyattsville: National Center for Health Statistics (US); 2017. Report No.: 2017-1232
8.• Fryar CD, Ostchega I, Hales CM, Zhang G, Kruszon-Moran D. Hypertension prevalence and control among adults: United States, 2015–2016. NCHS Data Brief. 2017;289:1–8. **This report shows the current prevalence of hypertension in US across sex and ethnic/racial groups.**
9. Lo JC, Sinaiko A, Chandra M, Daley MF, Greenspan LC, Parker ED, et al. Prehypertension and hypertension in community-based pediatric practice. Pediatrics. 2013;131(2):e415–e24.
10. Flack JM, Sica DA, Bakris G, Brown AL, Ferdinand KC, Grimm RH, et al. Management of high blood pressure in Blacks: an update of the International Society on Hypertension in Blacks consensus statement. Hypertension. 2010;56(5):780–800.

11. Kokubo Y, Iwashima Y. Higher blood pressure as a risk factor for diseases other than stroke and ischemic heart disease. Hypertension. 2015;66(2):254–9.
12. Ford ES, Cunningham TJ, Croft JB. Trends in self-reported sleep duration among US adults from 1985 to 2012. Sleep. 2015;38(5): 829–32.
13. Liu Y, Wheaton AG, Chapman DP, Cunningham TJ, Lu H, Croft JB. Prevalence of healthy sleep duration among adults—United States, 2014. MMWR Morb Mortal Wkly Rep. 2016;65:137–41.
14. Hirshkowitz M, Whiton K, Albert SM, Alessi C, Bruni O, DonCarlos L, et al. National Sleep Foundation's sleep time duration recommendations: methodology and results summary. Sleep Health. 2015;1(1):40–3.
15. Panel CC, Watson NF, Badr MS, Belenky G, Bliwise DL, Buxton OM, et al. Joint consensus statement of the American Academy of Sleep Medicine and Sleep Research Society on the recommended amount of sleep for a healthy adult: methodology and discussion. Sleep. 2015;38(8):1161–83.
16.• St-Onge M-P, Grandner MA, Brown D, Conroy MB, Jean-Louis G, Coons M, et al. Sleep duration and quality: impact on lifestyle behaviors and cardiometabolic health: a scientific statement from the American Heart Association. Circulation. 2016;134(18): e367–e86. **This statement reviews the evidence linking inadequate sleep quantity and quality to cardiometabolic risk and identifies research priorities in this area.**
17. Covassin N, Singh P. Sleep duration and cardiovascular disease risk: epidemiologic and experimental evidence. Sleep Med Clin. 2016;11(1):81–9.
18. Jackson CL, Redline S, Kawachi I, Hu FB. Association between sleep duration and diabetes in black and white adults. Diabetes Care. 2013;36(11):3557–65.
19. Cunningham TJ, Wheaton AG, Ford ES, Croft JB. Racial/ethnic disparities in self-reported short sleep duration among US-born and foreign-born adults. Ethn Health. 2016;21(6):628–38.
20.• Jean-Louis G, Grandner MA, Youngstedt SD, Williams NJ, Zizi F, Sarpong DF, et al. Differential increase in prevalence estimates of inadequate sleep among black and white Americans. BMC Public Health. 2015;15(1):1185. **This study shows the trajectories of sleep duration over time in Black and White individuals.**
21. Kim Y, Wilkens LR, Schembre SM, Henderson BE, Kolonel LN, Goodman MT. Insufficient and excessive amounts of sleep increase the risk of premature death from cardiovascular and other diseases: the Multiethnic Cohort Study. Prev Med. 2013;57(4): 377–85.
22. Malone SK, Patterson F, Lozano A, Hanlon A. Differences in morning–evening type and sleep duration between Black and White adults: results from a propensity-matched UK Biobank sample. Chronobiol Int. 2017;34(6):740–52.
23. National Sleep Foundation. 2010 Sleep in America poll: sleep and ethnicity. Washington, DC; National Sleep Foundation, 2010.
24. Whinnery J, Jackson N, Rattanaumpawan P, Grandner MA. Short and long sleep duration associated with race/ethnicity, sociodemographics, and socioeconomic position. Sleep. 2014;37(3):601–11.
25.• Stamatakis KA, Kaplan GA, Roberts RE. Short sleep duration across income, education, and race/ethnic groups: population prevalence and growing disparities during 34 years of follow-up. Ann Epidemiol. 2007;17(12):948–55. **This analysis of the Alameda County Health and Ways of Living Study reports on racial differences in prevalence and incidence of short sleep.**
26. Maslowsky J, Ozer EJ. Developmental trends in sleep duration in adolescence and young adulthood: evidence from a national United States sample. J Adolesc Health. 2014;54(6):691–7.
27. Chen X, Gelaye B, Williams MA. Sleep characteristics and health-related quality of life among a national sample of American young adults: assessment of possible health disparities. Qual Life Res. 2014;23(2):613–25.
28. Chen X, Wang R, Zee P, Lutsey PL, Javaheri S, Alcántara C, et al. Racial/ethnic differences in sleep disturbances: the Multi-Ethnic Study of Atherosclerosis (MESA). Sleep. 2015;38(6):877–88.
29. Lauderdale DS, Knutson KL, Yan LL, Rathouz PJ, Hulley SB, Sidney S, et al. Objectively measured sleep characteristics among early-middle-aged adults: the CARDIA study. Am J Epidemiol. 2006;164(1):5–16.
30. Lauderdale DS, Knutson KL, Yan LL, Liu K, Rathouz PJ. Self-reported and measured sleep duration: how similar are they? Epidemiology. 2008;19(6):838–45.
31. Fuller-Rowell TE, Curtis DS, El-Sheikh M, Chae DH, Boylan JM, Ryff CD. Racial disparities in sleep: the role of neighborhood disadvantage. Sleep Med. 2016;27:1–8.
32. Fuller-Rowell TE, Curtis DS, El-Sheikh M, Duke AM, Ryff CD, Zgierska AE. Racial discrimination mediates race differences in sleep problems: a longitudinal analysis. Cult Divers Ethn Minor Psychol. 2017;23(2):165.
33. Peterson LM, Miller KG, Wong PM, Anderson BP, Kamarck TW, Matthews KA, et al. Sleep duration partially accounts for race differences in diurnal cortisol dynamics. Health Psychol. 2017;36(5):502.
34.• Curtis DS, Fuller-Rowell TE, El-Sheikh M, Carnethon MR, Ryff CD. Habitual sleep as a contributor to racial differences in cardiometabolic risk. Proc Natl Acad Sci U S A. 2017;114(33):8889–94. **This study shows that sleep duration accounts for a significant portion of racial disparities in cardiometabolic risk markers including blood pressure.**
35. Mezick EJ, Matthews KA, Hall M, Strollo PJ Jr, Buysse DJ, Kamarck TW, et al. Influence of race and socioeconomic status on sleep: Pittsburgh Sleep SCORE project. Psychosom Med. 2008;70(4):410–6.
36. Hume KI, Brink M, Basner M. Effects of environmental noise on sleep. Noise Health. 2012;14(61):297–302.
37. Cho JR, Joo EY, Koo DL, Hong SB. Let there be no light: the effect of bedside light on sleep quality and background electroencephalographic rhythms. Sleep Med. 2013;14(12):1422–5.
38. Gold D, Leary P, Szpiro A, Aaron C, Kaufman J, Redline S, et al. Relationship of air pollution to sleep disruption: the Multi-Ethnic Study of Atherosclerosis (MESA) Sleep and MESA-Air Studies. Am J Respir Crit Care Med. 2017;195:A2930.
39. DeSantis AS, Diez Roux AV, Moore K, Baron KG, Mujahid MS, Nieto FJ. Associations of neighborhood characteristics with sleep timing and quality: the Multi-Ethnic Study of Atherosclerosis. Sleep. 2013;36(10):1543–51.
40. Pabayo R, Molnar BE, Street N, Kawachi I. The relationship between social fragmentation and sleep among adolescents living in Boston, Massachusetts. J Public Health. 2014;36(4):587–98.
41. Cunningham TJ, Ford ES, Chapman DP, Liu Y, Croft JB. Independent and joint associations of race/ethnicity and educational attainment with sleep-related symptoms in a population-based US sample. Prev Med. 2015;77:99–105.
42. Jackson CL, Redline S, Kawachi I, Williams MA, Hu FB. Racial disparities in short sleep duration by occupation and industry. Am J Epidemiol. 2013;178(9):1442–51.
43. Airhihenbuwa CO, Iwelunmor JI, Ezepue CJ, Williams NJ, Jean-Louis G. I sleep, because we sleep: a synthesis on the role of culture in sleep behavior research. Sleep Med. 2016;18:67–73.
44. Lewis TT, Troxel WM, Kravitz HM, Bromberger JT, Matthews KA, Hall MH. Chronic exposure to everyday discrimination and sleep in a multiethnic sample of middle-aged women. Health Psychol. 2013;32(7):810.
45. Åkerstedt T. Psychosocial stress and impaired sleep. Scand J Work Environ Health. 2006;32(6):493–501.

46. Johnson DA, Lisabeth L, Lewis TT, Sims M, Hickson DA, Samdarshi T, et al. The contribution of psychosocial stressors to sleep among African Americans in the Jackson Heart Study. Sleep. 2016;39(7):1411–9.
47. Grandner MA, Chakravorty S, Perlis ML, Oliver L, Gurubhagavatula I. Habitual sleep duration associated with self-reported and objectively determined cardiometabolic risk factors. Sleep Med. 2014;15(1):42–50.
48. Grandner MA, Jackson N, Gerstner JR, Knutson KL. Dietary nutrients associated with short and long sleep duration. Data from a nationally representative sample. Appetite. 2013;64:71–80.
49. Patterson F, Malone SK, Lozano A, Grandner MA, Hanlon AL. Smoking, screen-based sedentary behavior, and diet associated with habitual sleep duration and chronotype: data from the UK Biobank. Ann Behav Med. 2016;50(5):715–26.
50. Durrence HH, Lichstein KL. The sleep of African Americans: a comparative review. Behav Sleep Med. 2006;4(1):29–44.
51. Dudley KA, Patel SR. Disparities and genetic risk factors in obstructive sleep apnea. Sleep Med. 2016;18:96–102.
52. Kalmbach DA, Pillai V, Arnedt JT, Drake CL. DSM-5 insomnia and short sleep: comorbidity landscape and racial disparities. Sleep. 2016;39(12):2101–11.
53. Ollila HM, Kettunen J, Pietiläinen O, Aho V, Silander K, Kronholm E, et al. Genome-wide association study of sleep duration in the Finnish population. J Sleep Res. 2014;23(6):609–18.
54. Cade BE, Gottlieb DJ, Lauderdale DS, Bennett DA, Buchman AS, Buxbaum SG, et al. Common variants in DRD2 are associated with sleep duration: the CARe consortium. Hum Mol Genet. 2016;25(1):167–79.
55. Gottlieb DJ, Hek K, Chen T, Watson NF, Eiriksdottir G, Byrne EM, et al. Novel loci associated with usual sleep duration: the CHARGE Consortium Genome-Wide Association Study. Mol Psychiatry. 2015;20(10):1232–9.
56. Jones SE, Tyrrell J, Wood AR, Beaumont RN, Ruth KS, Tuke MA, et al. Genome-wide association analyses in 128,266 individuals identifies new morningness and sleep duration loci. PLoS Genet. 2016;12(8):e1006125.
57. Wu Y, Zhai L, Zhang D. Sleep duration and obesity among adults: a meta-analysis of prospective studies. Sleep Med. 2014;15(12):1456–62.
58. Shan Z, Ma H, Xie M, Yan P, Guo Y, Bao W, et al. Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. Diabetes Care. 2015;38(3):529–37.
59. Liu T-Z, Xu C, Rota M, Cai H, Zhang C, Shi M-J, et al. Sleep duration and risk of all-cause mortality: a flexible, non-linear, meta-regression of 40 prospective cohort studies. Sleep Med Rev. 2017;32:28–36.
60. Itani O, Jike M, Watanabe N, Kaneita Y. Short sleep duration and health outcomes: a systematic review, meta-analysis, and meta-regression. Sleep Med. 2017;32:246–56.
61. Gottlieb DJ, Redline S, Nieto FJ, Baldwin CM, Newman AB, Resnick HE, et al. Association of usual sleep duration with hypertension: the Sleep Heart Health Study. Sleep. 2006;29(8):1009–14.
62. Fang J, Wheaton AG, Keenan NL, Greenlund KJ, Perry GS, Croft JB. Association of sleep duration and hypertension among US adults varies by age and sex. Am J Hypertens. 2012;25(3):335–41.
63. Kim J, Jo I. Age-dependent association between sleep duration and hypertension in the adult Korean population. Am J Hypertens. 2010;23(12):1286–91.
64. Meng L, Zheng Y, Hui R. The relationship of sleep duration and insomnia to risk of hypertension incidence: a meta-analysis of prospective cohort studies. Hypertens Res. 2013;36(11):985–95.
65. Knutson KL, Van Cauter E, Rathouz PJ, Yan LL, Hulley SB, Liu K, et al. Association between sleep and blood pressure in midlife: the CARDIA sleep study. Arch Intern Med. 2009;169(11):1055–61.
66.• Guo X, Zheng L, Wang J, Zhang X, Zhang X, Li J, et al. Epidemiological evidence for the link between sleep duration and high blood pressure: a systematic review and meta-analysis. Sleep Med. 2013;14(4):324–32. **This recent meta-analysis provides pooled estimates on the cross-sectional and longitudinal associations between inadequate sleep and hypertension.**
67. Wang Q, Xi B, Liu M, Zhang Y, Fu M. Short sleep duration is associated with hypertension risk among adults: a systematic review and meta-analysis. Hypertens Res. 2012;35(10):1012–8.
68.• Pandey A, Williams N, Donat M, Ceide M, Brimah P, Ogedegbe G, et al. Linking sleep to hypertension: greater risk for blacks. Int J Hypertens. 2013;2013:436502. **This analysis of a nationwide survey shows that the prevalence of hypertension in Black short sleepers is higher than in their White counterparts.**
69. Rogers A, Necola O, Sexias A, Luka A, Newsome V, Williams S, et al. Resistant hypertension and sleep duration among blacks with metabolic syndrome MetSO. J Sleep Disord Treat Care. 2016;5(4)
70. Grandner MA, Buxton OM, Jackson N, Sands-Lincoln M, Pandey A, Jean-Louis G. Extreme sleep durations and increased C-reactive protein: effects of sex and ethnoracial group. Sleep. 2013;36(5):769–79.
71. Jean-Louis G, Youngstedt S, Grandner M, Williams NJ, Sarpong D, Zizi F, et al. Unequal burden of sleep-related obesity among black and white Americans. Sleep Health. 2015;1(3):169–76.
72. Donat M, Brown C, Williams N, Pandey A, Racine C, McFarlane SI, et al. Linking sleep duration and obesity among black and white US adults. Clin Pract (Lond). 2013;10(5):661–7.
73. Hill A, Williams N, Salifu I, Castor C, Gibilaro J. The role of race/ethnicity and gender in the association between inadequate sleep and hypercholesterolemia. J Sleep Disord Ther. 2015;4(194):1000194.
74. Cai A, Wang L, Zhou Y. Hypertension and obstructive sleep apnea. Hypertens Res. 2016;39(6):391–5.
75.• Ancoli-Israel S, Stepnowsky C, Dimsdale J, Marler M, Cohen-Zion M, Johnson S. The effect of race and sleep-disordered breathing on nocturnal BP "dipping": analysis in an older population. Chest. 2002;122(4):1148–55. **This study reports that non-dipping is more common in African-Americans than in Whites irrespective of comorbid sleep-disordered breathing.**
76. 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(9):982–8.
77. Kato M, Phillips BG, Sigurdsson G, Narkiewicz K, Pesek CA, Somers VK. Effects of sleep deprivation on neural circulatory control. Hypertension. 2000;35(5):1173–5.
78. Carter JR, Durocher JJ, Larson RA, DellaValla JP, Yang H. Sympathetic neural responses to 24-hour sleep deprivation in humans: sex differences. Am J Physiol Heart Circ Physiol. 2012;302(10):H1991–7.
79. Ogawa Y, Kanbayashi T, Saito Y, Takahashi Y, Kitajima T, Takahashi K, et al. Total sleep deprivation elevates blood pressure through arterial baroreflex resetting: a study with microneurographic technique. Sleep. 2003;26(8):986–9.
80. Sauvet F, Leftheriotis G, Gomez-Merino D, Langrume C, Drogou C, Van Beers P, et al. Effect of acute sleep deprivation on vascular function in healthy subjects. J Appl Physiol. 2010;108(1):68–75.
81. Sunbul M, Kanar BG, Durmus E, Kivrak T, Sari I. Acute sleep deprivation is associated with increased arterial stiffness in healthy young adults. Sleep Breath. 2014;18(1):215–20.
82. Meier-Ewert HK, Ridker PM, Rifai N, Regan MM, Price NJ, Dinges DF, et al. Effect of sleep loss on C-reactive protein, an inflammatory marker of cardiovascular risk. J Am Coll Cardiol. 2004;43(4):678–83.

83. Dettoni JL, Consolim-Colombo FM, Drager LF, Rubira MC, de Souza SBPC, Irigoyen MC, et al. Cardiovascular effects of partial sleep deprivation in healthy volunteers. J Appl Physiol. 2012;113(2):232–6.
84. van Leeuwen WM, Sallinen M, Virkkala J, Lindholm H, Hirvonen A, Hublin C, et al. Physiological and autonomic stress responses after prolonged sleep restriction and subsequent recovery sleep in healthy young men. Sleep Biol Rhythm. 2018;16(1):45–54.
85. Grimaldi D, Carter JR, Van Cauter E, Leproult R. Adverse impact of sleep restriction and circadian misalignment on autonomic function in healthy young adults. Hypertension. 2016;68(1):243–50.
86. Choudhary AK, Dadarao Dhanvijay AK, Alam T, Kishanrao SS. Sleep restriction and its influence on blood pressure. Artery Res. 2017;19:42–8.
87.• Yang H, Haack M, Gautam S, Meier-Ewert HK, Mullington JM. Repetitive exposure to shortened sleep leads to blunted sleep-associated blood pressure dipping. J Hypertens. 2017;35(6): 1187–94. **This experimental study illustrates the effects of repeated cycles of sleep restriction on diurnal blood pressure dynamics.**
88. Broussard JL, Chapotot F, Abraham V, Day A, Delebecque F, Whitmore HR, et al. Sleep restriction increases free fatty acids in healthy men. Diabetologia. 2015;58(4):791–8.
89. Faraut B, Nakib S, Drogou C, Elbaz M, Sauvet F, De Bandt JP, et al. Napping reverses the salivary interleukin-6 and urinary norepinephrine changes induced by sleep restriction. J Clin Endocrinol Metab. 2015;100(3):E416–26.
90. Nedeltcheva AV, Kessler L, Imperial J, Penev PD. Exposure to recurrent sleep restriction in the setting of high caloric intake and physical inactivity results in increased insulin resistance and reduced glucose tolerance. J Clin Endocrinol Metab. 2009;94(9): 3242–50.
91. James JE, Gregg ME. Hemodynamic effects of dietary caffeine, sleep restriction, and laboratory stress. Psychophysiology. 2004;41(6):914–23.
92. Calvin AD, Covassin N, Kremers WK, Adachi T, Macedo P, Albuquerque FN, et al. Experimental sleep restriction causes endothelial dysfunction in healthy humans. J Am Heart Assoc. 2014;3(6):e001143.
93. Sauvet F, Drogou C, Bougard C, Arnal PJ, Dispersyn G, Bourrilhon C, et al. Vascular response to 1 week of sleep restriction in healthy subjects. A metabolic response? Int J Cardiol. 2015;190:246–55.
94. Charloux A, Gronfier C, Chapotot F, Ehrhart J, Piquard F, Brandenberger G. Sleep deprivation blunts the night time increase in aldosterone release in humans. J Sleep Res. 2001;10(1):27–33.
95. Kamperis K, Hagstroem S, Radvanska E, Rittig S, Djurhuus JC. Excess diuresis and natriuresis during acute sleep deprivation in healthy adults. Am J Physiol Renal Physiol. 2010;299(2):F404–11.
96. Chida Y, Steptoe A. Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status. Hypertension. 2010;55(4):1026–32.
97. Lagraauw HM, Kuiper J, Bot I. Acute and chronic psychological stress as risk factors for cardiovascular disease: insights gained from epidemiological, clinical and experimental studies. Brain Behav Immun. 2015;50:18–30.
98. Treiber FA, Kamarck T, Schneiderman N, Sheffield D, Kapuku G, Taylor T. Cardiovascular reactivity and development of preclinical and clinical disease states. Psychosom Med. 2003;65(1):46–62.
99. Franzen PL, Gianaros PJ, Marsland AL, Hall MH, Siegle GJ, Dahl RE, et al. Cardiovascular reactivity to acute psychological stress following sleep deprivation. Psychosom Med. 2011;73(8):679–82.
100. Yang H, Durocher JJ, Larson RA, DellaValla JP, Carter JR. Total sleep deprivation alters cardiovascular reactivity to acute stressors in humans. J Appl Physiol. 2012;113(6):903–8.
101. Minkel J, Moreta M, Muto J, Htaik O, Jones C, Basner M, et al. Sleep deprivation potentiates HPA axis stress reactivity in healthy adults. Health Psychol. 2014;33(11):1430–4.
102.• Muntner P, Lewis CE, Diaz KM, Carson AP, Kim Y, Calhoun D, et al. Racial differences in abnormal ambulatory blood pressure monitoring measures: results from the Coronary Artery Risk Development in Young Adults (CARDIA) study. Am J Hypertens. 2014;28(5):640–8. **This cross-sectional examination of the CARDIA study shows that racial-dependent differences in ambulatory blood pressure patterns are amplified during nighttime.**
103. Jehn ML, Brotman DJ, Appel LJ. Racial differences in diurnal blood pressure and heart rate patterns: results from the Dietary Approaches to Stop Hypertension (DASH) trial. Arch Intern Med. 2008;168(9):996–1002.
104. Viera AJ, Zhu S, Hinderliter AL, Shimbo D, Person SD, Jacobs DR Jr. Diurnal blood pressure pattern and development of prehypertension or hypertension in young adults: the CARDIA study. J Am Soc Hypertens. 2011;5(1):48–55.
105. Holwerda SW, Fulton D, Eubank WL, Keller DM. Carotid baroreflex responsiveness is impaired in normotensive African American men. Am J Physiol Heart Circ Physiol. 2011;301(4): H1639–45.
106. Adefurin A, Ghimire LV, Kohli U, Muszkat M, Sofowora GG, Paranjape SY, et al. Blacks have a greater sensitivity to α1-adrenoceptor-mediated venoconstriction compared with Whites. Hypertension. 2013;61(4):915–20.
107. Sherwood A, Hill LK, Blumenthal JA, Johnson KS, Hinderliter AL. Race and sex differences in cardiovascular α-adrenergic and β-adrenergic receptor responsiveness in men and women with high blood pressure. J Hypertens. 2017;35(5):975–81.
108. Stein CM, Lang CC, Singh I, He HB, Wood AJ. Increased vascular adrenergic vasoconstriction and decreased vasodilation in blacks. Hypertension. 2000;36(6):945–51.
109. Ray CA, Monahan KD. Sympathetic vascular transduction is augmented in young normotensive blacks. J Appl Physiol. 2002;92(2):651–6.
110. Sherwood A, Steffen PR, Blumenthal JA, Kuhn C, Hinderliter AL. Nighttime blood pressure dipping: the role of the sympathetic nervous system. Am J Hypertens. 2002;15(2):111–8.
111. Patel PD, Velazquez JL, Arora RR. Endothelial dysfunction in African-Americans. Int J Cardiol. 2009;132(2):157–72.
112. Morris AA, Patel RS, Binongo JNG, Poole J, al Mheid I, Ahmed Y, et al. Racial differences in arterial stiffness and microcirculatory function between black and white Americans. J Am Heart Assoc. 2013;2(2):e002154.
113. Hill LK, Sherwood A, Blumenthal JA, Hinderliter AL. Hemodynamics and vascular hypertrophy in African Americans and Caucasians with high blood pressure. Am J Hypertens. 2016;29(12):1380–5.
114. Taherzadeh Z, Brewster LM, Van Montfrans GA, VanBavel E. Function and structure of resistance vessels in black and white people. J Clin Hypertens (Greenwich). 2010;12(6):431–8.
115. Williams SF, Nicholas SB, Vaziri ND, Norris KC. African Americans, hypertension and the renin angiotensin system. World J Cardiol. 2014;6(9):878–89.
116. Elijovich F, Weinberger MH, Anderson CA, Appel LJ, Bursztyn M, Cook NR, et al. Salt sensitivity of blood pressure: a scientific statement from the American Heart Association. Hypertension. 2016;68(3):e7–46.
117. Hamer M, Malan L. Psychophysiological risk markers of cardiovascular disease. Neurosci Biobehav Rev. 2010;35(1):76–83.

118. Murphy JK, Alpert BS, Walker SS. Ethnicity, pressor reactivity, and children's blood pressure. Five years of observations. Hypertension. 1992;20(3):327–32.
119. Knox SS, Hausdorff J, Markovitz JH. Reactivity as a predictor of subsequent blood pressure: racial differences in the Coronary Artery Risk Development in Young Adults (CARDIA) study. Hypertension. 2002;40(6):914–9.
120. McEwen BS, Karatsoreos IN. Sleep deprivation and circadian disruption: stress, allostasis, and allostatic load. Sleep Med Clin. 2015;10(1):1–10.
121. Burford NG, Webster NA, Cruz-Topete D. Hypothalamic-pituitary-adrenal axis modulation of glucocorticoids in the cardiovascular system. Int J Mol Sci. 2017;18(10):E2150.
122. Barksdale DJ, Woods-Giscombé C, Logan JG. Stress, cortisol, and nighttime blood pressure dipping in nonhypertensive black American women. Biol Res Nurs. 2013;15(3):330–7.
123. Buxton OM, Pavlova M, Reid EW, Wang W, Simonson DC, Adler GK. Sleep restriction for 1 week reduces insulin sensitivity in healthy men. Diabetes. 2010;59(9):2126–33.
124. Broussard JL, Ehrmann DA, Van Cauter E, Tasali E, Brady MJ. Impaired insulin signaling in human adipocytes after experimental sleep restriction: a randomized, crossover study. Ann Intern Med. 2012;157(8):549–57.
125. Reynolds AC, Dorrian J, Liu PY, Van Dongen HPA, Wittert GA, Harmer LJ, et al. Impact of five nights of sleep restriction on glucose metabolism, leptin and testosterone in young adult men. PLoS One. 2012;7(7):e41218.
126. Markwald RR, Melanson EL, Smith MR, Higgins J, Perreault L, Eckel RH, et al. Impact of insufficient sleep on total daily energy expenditure, food intake, and weight gain. Proc Natl Acad Sci U S A. 2013;110(14):5695–700.
127. Simpson NS, Banks S, Dinges DF. Sleep restriction is associated with increased morning plasma leptin concentrations, especially in women. Biol Res Nurs. 2010;12(1):47–53.
128. Calvin AD, Carter RE, Adachi T, Macedo PG, Albuquerque FN, van der Walt C, et al. Effects of experimental sleep restriction on caloric intake and activity energy expenditure. Chest. 2013;144(1):79–86.
129.• Spaeth AM, Dinges DF, Goel N. Effects of experimental sleep restriction on weight gain, caloric intake, and meal timing in healthy adults. Sleep. 2013;36(7):981–90. **This experimental study reports that African-Americans exposed to in-laboratory sleep restriction gain more weight than Whites.**
130. Guyon A, Balbo M, Morselli LL, Tasali E, Leproult R, L'Hermite-Baleriaux M, et al. Adverse effects of two nights of sleep restriction on the hypothalamic-pituitary-adrenal axis in healthy men. J Clin Endocrinol Metab. 2014;99(8):2861–8.
131. van Leeuwen WM, Lehto M, Karisola P, Lindholm H, Luukkonen R, Sallinen M, et al. Sleep restriction increases the risk of developing cardiovascular diseases by augmenting proinflammatory responses through IL-17 and CRP. PLoS One. 2009;4(2):e4589.
132.• Spaeth AM, Dinges DF, Goel N. Sex and race differences in caloric intake during sleep restriction in healthy adults. Am J Clin Nutr. 2014;100(2):559–66. **This study presents the effects of experimentally-induced sleep restrion on calorie intake and reports differences on nutritional choices between African-American and White subjects.**
133. Saab KR, Kendrick J, Yracheta JM, Lanaspa MA, Pollard M, Johnson RJ. New insights on the risk for cardiovascular disease in African Americans: the role of added sugars. J Am Soc Nephrol. 2015;26(2):247–57.
134. Stamler J, Brown IJ, Yap IK, Chan Q, Wijeyesekera A, Garcia-Perez I, et al. Dietary and urinary metabonomic factors possibly accounting for higher blood pressure of black compared with white Americans: results of International Collaborative Study on macro-/micronutrients and blood pressure. Hypertension. 2013;62(6):1074–80.
135. Svetkey LP, Simons-Morton D, Vollmer WM, Appel LJ, Conlin PR, Ryan DH, et al. Effects of dietary patterns on blood pressure: subgroup analysis of the Dietary Approaches to Stop Hypertension (DASH) randomized clinical trial. Arch Intern Med. 1999;159(3):285–93.
136. Cogswell ME, Zhang Z, Carriquiry AL, Gunn JP, Kuklina EV, Saydah SH, et al. Sodium and potassium intakes among US adults: NHANES 2003–2008. Am J Clin Nutr. 2012;96(3):647–57.
137. Bell BB, Rahmouni K. Leptin as a mediator of obesity-induced hypertension. Curr Obes Rep. 2016;5(4):397–404.
138. Kim DH, Kim C, Ding EL, Townsend MK, Lipsitz LA. Adiponectin levels and the risk of hypertension: a systematic review and meta-analysis. Hypertension. 2013;62(1):27–32.
139. Simpson NS, Banks S, Arroyo S, Dinges DF. Effects of sleep restriction on adiponectin levels in healthy men and women. Physiol Behav. 2010;101(5):693–8.