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Sleep and its Relationship to Racial and Ethnic Disparities in Cardiovascular Disease

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Abstract

There are substantial racial/ethnic disparities in cardiovascular disease in the U.S., but few mechanisms have emerged as feasible intervention targets. A growing body of research suggests that racial/ethnic differences in sleep deficiency, including extreme sleep duration, sleep-disordered breathing, and insomnia, may help explain disparities in cardiovascular disease. However, little is known about the mechanisms underlying racial/ethnic disparities in sleep. In this article, we review the extant literature on sleep and cardiovascular outcomes (e.g., hypertension, stroke, cardiovascular disease) and racial/ethnic differences in these relations. We also discuss possible mechanisms that might help explain racial/ethnic sleep disparities, including neighborhood disadvantage, psychosocial and occupational stressors, acculturation, and treatment access and adherence. More research is needed to establish causal linkages among race/ethnicity, sleep, and these mechanisms, but existing evidence suggests that targeting these factors in interventions may reduce racial/ethnic sleep disparities and improve primary prevention of cardiovascular disease among all racial/ethnic groups.

Keywords

sleep; sleep duration; sleep-disordered breathing; insomnia; race; ethnicity; disparities; cardiovascular disease

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Racial/ethnic disparities in cardiovascular disease (CVD) pose a serious public health problem in the U.S. Relative to non-Hispanic Whites, African Americans/Blacks are 43% more likely to develop hypertension, 95% more likely to have a stroke, and 30% more likely to die from CVD.(1) There are many potential mechanisms that likely contribute to CVD disparities (e.g., social and physical environment), but few have emerged as feasible intervention targets. Sleep disturbances and sleep disorders are disproportionately prevalent in minority groups. Therefore, sleep may help explain CVD disparities and provide novel targets to improve CV health.(2) We review the literature on the interplay among race/ethnicity, sleep duration, two common sleep disorders (sleep-disordered breathing [SDB] and insomnia), and CVD, highlighting mechanisms that may explain disparities.

What is Sleep's Purpose?

Sleep is a distinct neurophysiological state during which characteristic physiological changes occur. Brain electrical and metabolic activities and autonomic nervous system activity vary with sleep versus wake states and across sleep stages. Variations in autonomic nervous system activity, hormone levels, and metabolism influence intermediate pathways to CVD, including diurnal patterns of blood pressure and heart rate, insulin sensitivity, and fluid and salt homeostasis. Sleep serves a restorative function for the brain and body, and is an integral part of overall health and a source of physiological and psychological resilience.

Humans have evolved to require 7-9 hours per night during adulthood, with absolute variation in sleep need likely influenced by genetic and other individual factors. There is evidence that sleep disorders are associated with increased risk of accidents, injuries,(3,4) and reduced health-related quality of life and cognitive functioning (5). Sleep deficiency, “a deficit in the quantity or quality of sleep obtained versus the amount needed for optimal health and well-being,” (6) has deleterious effects on the autonomic nervous system, and thus, is particularly stressful on the CV system (see Figure 1).(7,8)

Sleep Duration and CVD

Extreme sleep duration

A review of 15 studies on sleep duration and CV outcomes found strong evidence that short sleep duration is associated with a greater risk of stroke and developing or dying from CVD. (9) Similarly, a systematic review of 30 studies found that self-reported short sleepers are 55% more likely to be obese than those reporting 7 hours of sleep per night.(10) National Health Interview Survey (NHIS) data indicate that, relative to sleeping 7-8 hours/night, self-reported short (< 6 hours) and long sleep (>9 hours) are independently associated with increased obesity, and diagnosis of diabetes, hypertension, and CVD.(11)

Mechanisms linking short sleep and CVD likely include perturbations of the hypothalamic-pituitary-adrenal axis as well as disturbances in autonomic activity. Experimental studies testing physiological effects of acute sleep deprivation observed increased diastolic blood pressure, abnormalities in muscle sympathetic nerve activity,(12) reduced leptin, and increased ghrelin levels,(13) elevated afternoon and evening cortisol, and reduced insulin sensitivity.(14) Prospective studies have linked short sleep duration with incident hypertension (15) and coronary artery calcification. (16)

Although long sleep (> 9 hours per night) has been linked with adverse CVD outcomes, the basis for this association is unclear. Long sleep may occur in various chronic psychiatric and medical conditions, which may be unmeasured confounders. Long sleep may also be associated with excessive sedentary behavior, which may independently influence CVD.

Furthermore, long sleep may be due to over-compensation for poor sleep quality stemming from a sleep disorder (e.g., SDB).(17)

Racial/ethnic differences in extreme sleep duration

Extremes of sleep duration vary across racial/ethnic groups. Compared to Whites, Blacks are nearly twice as likely to report short sleep,(18,19), and over 60% more likely to report long sleep.(20) One study estimated that Blacks sleep roughly 35-60 minutes less per night than do Whites.(21-23) A recent meta-analysis of 14 studies found that Blacks were more likely to be short sleepers than were Whites, with larger effect sizes in studies using objective as compared to. self-reported measures of sleep duration.(24) Self-report measures may underestimate the magnitude of racial differences in sleep duration.

Blacks may also be at risk for more severe consequences of extreme sleep than Whites. Analysis of NHIS data revealed that, among individuals reporting short or long sleep duration, Blacks were at greater risk of diabetes than were Whites.(25) Moreover, these effects remained significant when controlling for age, sex, and income.

Less research has examined sleep health in Hispanics, but existing evidence suggests that they, too, may be at increased risk for extreme sleep duration.(26) Relative to non-Hispanic whites, Hispanics are more likely to report short (20) and long sleep duration,(19) controlling for confounders. More work is needed to investigate whether similar effects are present among other minority groups, and for other CV outcomes.

In sum, extreme sleep durations are disproportionately common in low-income and minority groups, and are associated with intermediate CVD mechanisms, and subclinical and clinical CVD. Although there are limited longitudinal data available, it appears likely that extreme sleep duration may mediate a portion of the increased CVD burden observed in African Americans and Hispanic groups.

Sleep Disorders and CVD

Sleep disorders, such as SDB and insomnia, also influence the pathogenesis of CVD.

Sleep-disordered breathing (SDB)

SDB is the occurrence of repetitive episodes of pharyngeal obstruction during sleep manifest as recurrent hypopneas or apneas. SDB is associated with symptoms of sleep disruption, snoring, and daytime sleepiness. The physiological effects of SDB-associated recurrent intermittent hypoxemia, intrathoracic pressure swings, brain arousals, and sympathetic nervous system activation adversely impact blood pressure, endothelial function, insulin sensitivity, atherogenesis, and cardiac function.(27-29) Epidemiological studies have established SDB as a risk factor for incident hypertension,(30) heart failure, coronary artery disease, and stroke.(31,32) SDB treatment with nasal continuous positive airway pressure (CPAP) improves insulin sensitivity and metabolic indices, decreases blood pressure (33,34) and reduces rates of fatal and non-fatal CVD (35) and incident hypertension.(30)

Racial/ethnic differences in SDB

Minority group members may be at greater risk for more frequent and severe presentations of SDB than Whites. Racial differences in SDB are most evident among children and young adults, as Black children and young adults are 4-6 times more likely to have SDB than their White counterparts, (37,38) with differences emerging very early.

A cross-sectional study of 346 children age 2-6 found that the odds of snoring were three times greater among Blacks than Whites.(36) Self-reported “frequent” snoring is also more

common among Hispanic and Black adults than among Whites.(37) One study showed that Hispanic and Black adults were nearly three times as likely to exhibit higher frequency of oxygen desaturation events during sleep as were Whites.(38) A cross-sectional study of 280 patients with obstructive sleep apnea found that: (a) relative to Whites, Blacks were significantly more obese and had higher rates of hypertension at the time of sleep apnea diagnosis, (b) Black females were diagnosed at a significantly younger age than were White females, and (c) Black males had significantly lower oxygen saturation level than did White males, controlling for BMI and age.(39) Thus, Blacks may experience earlier and more severe presentations of SDB than Whites, and SDB may contribute to a lifelong increased risk of CVD among minority groups.

Insomnia

Insomnia is defined as difficulty falling or remaining asleep, despite adequate opportunities to sleep, with associated daytime impairment. It reflects a condition of cognitive and/or physiological “hyperarousal,” as evidenced by its associations with worry and psychological stress, and alterations in metabolic rate, heart rate variability, and cortisol secretion.(40-43) For example, in a study of over 4300 Danish civil servants, insomnia symptoms were associated with reduced cortisol awakening response and a flattened diurnal cortisol profile.(44) Given these pathophysiological correlates, it is not surprising that there is a growing literature linking insomnia to CV morbidity. Laugsand and colleagues found that insomnia symptoms were associated with greater risk of acute myocardial infarction in a study of >50,000 adults.(45) A recent review estimated that the presence of insomnia symptoms is associated with a relative risk of CVD ranging from 1.5-3.9.(46) Recent evidence suggests that the link between insomnia and CVD may be especially strong among short sleepers. A prospective study of nearly 1400 adults found that the risk of incident hypertension was highest among those with self-reported chronic insomnia *and* objective short sleep duration, controlling for SDB.(47) Together, these studies indicate a robust link between insomnia and CV morbidity. Further research is needed to dissect the associations among sleep duration, insomnia, and CVD.

Racial/ethnic differences in insomnia

Racial differences in insomnia are not well-understood.(20) One cross-sectional study of 1741 adults found that self-reported chronic insomnia (i.e., symptoms >1 year) was more common among minority group members than among Whites.(48) Similarly, a survey of 92 primary care patients found that Blacks were more likely than Whites to report sleep disturbance.(49) This relationship was partially attenuated when controlling for SES, depression, and medical illness. Similar results were found in a prospective study: non-Whites suffered from more chronic insomnia, controlling for potential confounders and baseline insomnia symptoms.(50)

In contrast, other studies have found that minorities are at *lower* risk of insomnia. A survey of over 17,000 adults revealed that Whites reported more trouble falling asleep and staying asleep than did Blacks and Hispanics.(51) Similar findings were reported in a diary study examining self-reported chronic insomnia in 769 adults.(52)

These mixed findings may be due, in part, to methodological issues, such as differing definitions of insomnia or reliance on self-reports. Of the studies reviewed here, those with the strongest methods (e.g., use of validated self-report measures, prospective designs) found evidence of a racial difference in insomnia;(49) still, more methodologically-sound studies are needed to establish the true nature of the link between race/ethnicity and insomnia, and better understand how perceived symptoms and physiological differences in sleep vary by ethnicity/race.

Possible Mechanisms (see Figure 2 for conceptual model)

There is considerable evidence linking extreme sleep duration, SDB, and insomnia with CVD markers and CV outcomes. Poor sleep health is associated with physiological responses (e.g., hormonal changes, increased blood pressure, reduced insulin sensitivity), which may have harmful downstream consequences for CV health. Because there is evidence of racial differences in both extreme sleep duration and SDB, and to a slightly lesser extent, insomnia, these factors have received consideration as contributors to observed ethno-racial disparities in CV outcomes. Less is known about the mediating mechanisms that explain *why* racial and ethnic minorities are at greater risk for sleep deficiency than are Whites.

Neighborhood disadvantage

One possible way in which race/ethnicity influences sleep is through increased exposure to various environmental physical and emotional stressors. For example, poor urban environments have higher levels of air pollution,(53,54) which may lead to an increased prevalence of SDB.(55) Other studies have found an association between neighborhood disorder and self-reported sleep quality.(56,57) Those living in poorer neighborhoods are more likely to be exposed to factors that may contribute to sleep deficiency, such as inopportune light exposure, noise, allergens, and irritants (e.g., environmental tobacco).(58,59) Some of these factors (such as particulate air pollution) may also contribute to abnormalities in the autonomic nervous system and increase CV morbidity. Because minority group members are more likely to live in disadvantaged neighborhoods than are Whites,(60) the adverse effects of stressors and exposures in disadvantaged neighborhoods may contribute to CVD disparities via an influence on sleep.

Occupational stressors

Minorities are also at greater risk of suffering from occupational stressors, which have been linked with sleep deficiency,(61) self-reported sleep quality, continuity,(62) and duration.(63) In the U.S., Blacks and Hispanics are more likely to work the night shift, less likely to have flexible work schedules, and more likely to work greater than 40 hours per week than are Whites.(23,64) Work-environment characteristics, such as harassment on the job and low supervisor support, are associated with increased sleep deficiency.(65) Having an inflexible manager with respect to work and family needs is associated with both shorter sleep duration on the order of one-half hour per day, and a doubling of CVD risk.(66) Shift work is associated with abnormalities in intermediate markers of CVD such as blood pressure and glucose levels, as well as with myocardial infarction, and diabetes.(67,68) Taken together, these studies suggest that minorities may suffer from greater sleep deficiency and CVD risk factors in part from increased exposure to occupational stressors that include shift work, long work hours,(23) inflexible work schedules, and possibly workplace discrimination. It is also likely that workplace exposures to airborne irritants may contribute to SDB.

Psychosocial stressors

Researchers have suggested that psychosocial factors should receive greater attention as contributors to health disparities. One of the primary psychosocial stressors that is disproportionately experienced by minorities, and may be implicated in sleep disparities, is perceived racial discrimination.(69,70) Reports of lifetime discrimination were positively associated with self-reported insufficient sleep in a survey of over 7000 adults.(71) Similarly, a study of 168 Hispanics found that perceived racism was predictive of self-reported sleep disturbance.(72) Perceived discrimination has also been shown to influence sleep architecture. As mentioned previously, Blacks have less slow wave sleep than do

Whites,(73) and this effect appears to be partially mediated by perceived racial discrimination.(74) More work is needed to unpack the effects of discrimination on sleep, but existing evidence suggests that the stress of discrimination influences minority group members' sleep quality and duration, and thus, may have downstream effects on CV outcomes.

Acculturation

Recent evidence suggests that the harmful effects of acculturation (75,76),(77)(78,79) among Hispanics extend to sleep duration. A nationally-representative study found that Mexican-Americans were 44% more likely to report short sleep duration than were Mexican immigrants, when controlling for confounders.(80) This effect was modestly attenuated with the addition of smoking and self-reported stress to the model, suggesting that these variables may help explain the link between acculturation and sleep duration. Acculturation has also been linked to sleep disturbance. A study of over 300 women of Mexican descent found that two measures of acculturation, language preference (Spanish or English) and socialization in the U.S. before age 18, were significant predictors of self-reported sleep disturbance.(81) These studies provide initial evidence highlighting acculturation as a mechanism by which Mexican-Americans experience greater sleep deficiency than Whites. Future studies should investigate this relation in other racial/ethnic groups and dissect the extent to which acculturation reflects the influences of a “Westernized” lifestyle or reflects stress associated with acculturation.

Treatment access and adherence

Limited access to treatment for sleep disorders has been linked with race/ethnicity, and may play a role in sleep and CVD disparities. Minority group members suffering from sleep disorders may receive later diagnoses, and have less access to treatment than Whites.(82) Moreover, there is evidence suggesting that, independent of *access* to treatment, *adherence* to sleep disorder treatments is lower among Blacks than Whites,(83,84) which may unnecessarily increase incidence and severity of CV outcomes. The HomePAP study, which provided standardized care to all participants, found that Blacks were less likely to adhere to CPAP treatment than were Whites, and this effect was partially mediated by self-reported short sleep duration.(85) Differences in socioeconomic resources, social support, and perceived benefits/risks of treatment may underlie racial differences in treatment adherence.(86) Therefore, increasing sleep duration among individuals suffering from SDB may help improve treatment adherence, and reduce disparities in CV outcomes.

Future Directions and Needed Research

Much research has investigated sleep and its relationship to racial and ethnic disparities in CVD, but many questions remain unanswered. Research is needed to investigate possible mechanisms by which race/ethnicity is related to sleep, and protective factors that could be targeted by interventions aimed at promoting healthy sleep among all groups.

Clearer definitions of “short” and “long” sleep are needed in studies investigating extreme sleep duration and CV outcomes. The lack of universal definitions for these terms poses problems for meta-analyses and reviews.(17) Moreover, more work is needed to assess individual differences in sleep need, which will help researchers better identify the presence of sleep deficiency due to insufficient sleep duration. Future studies should employ more objective measures of sleep, such as actigraphy and polysomnography, or survey measures that have been validated against objective assessments in each group of interest. These methodological improvements are critical for determining a more accurate estimate of the effects of sleep on CV outcomes across different racial/ethnic groups.

More research is also needed to examine the possible bidirectional relationships between stress and sleep, and their associations with CV outcomes. Prospective studies will be critical in establishing temporal ordering of sleep duration and sleep problems, stress, and CV outcomes for different racial/ethnic groups. In addition, more work is needed to investigate how the nature of stressors influences sleep. Stressors can take many different forms, including those that are sudden, daily, and chronic.(87) Examining how the nature of stressors differs by race/ethnicity to predict sleep health could provide insight into sleep disparities. Studies are also needed to identify key demographic, personality, environmental, and genetic moderators of the effects of race on sleep, and sleep on CVD, and to better understand whether sleep disturbances differentially contribute to CVD risk in individuals of different ethnic/racial backgrounds. For example, shift workers may be at especially high risk for CVD if they also live in a poor, urban neighborhood; or, the effects of perceived discrimination on sleep may be mitigated for individuals who possess a strong sense of racial/ethnic identity. Research examining potential genetic and environmental moderators would provide a more critical understanding of the mechanistic and possible epigenetic links among race, sleep, and CV outcomes.

Finally, improvements to housing policy and culturally-relevant intervention efforts are needed to improve the sleep habits and environment of all racial/ethnic groups. For example, policies to promote safe neighborhoods, such as increased nocturnal street light, may inadvertently create barriers to healthy sleep. Moreover, subsidized housing complexes may not provide residents with the necessary resources to establish a healthy sleep environment (e.g., temperature controls, window shading). Housing policy should include measures that require new complexes to: (a) be built away from noisy and polluted roadways; (b) be constructed so as to minimize internal noise; (c) provide heating and air conditioning control; and (d) come equipped with blinds that sufficiently block outside light.

At an individual level, intervention programs should provide education about ways to improve the home sleep environment, such as the importance of a quiet, comfortable bedroom and shutting off the television and other devices when trying to fall asleep, and of the importance of sleep to overall health. Previous education-based interventions have proven effective at improving sleep hygiene and quality in university populations.(88) Similar interventions should be tailored to minority populations and integrated within programs for primary or secondary CVD risk prevention.

Conclusion: Implications for CVD Prevention and Management

Sleep is an integral part of overall health, particularly cardiovascular health. Sleep deficiencies are linked with numerous negative health outcomes including obesity, hypertension, and diabetes. Racial and ethnic minorities tend to experience more extreme sleep duration and sleep disorders than do Whites. These disparities in sleep health may contribute to the disproportionately high rates of CVD among minorities. Research investigating the mechanisms underlying these effects is in its early stages, but there is evidence suggesting that greater exposure to stressors (e.g., neighborhood, occupational, psychosocial), acculturation, and relatively low treatment access and adherence may help explain racial differences in sleep deficiency. More work is needed to identify protective factors that could be targeted by culturally-relevant interventions. Such interventions would help promote more positive sleep health among minorities, and thus, help reduce racial/ethnic disparities in CVD. However, the existing data support the need to address sleep disorders as a means for primary and secondary cardiovascular disease reduction.

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Age-adjusted percentage of adults who reported 30 days of insufficient rest or sleep during the preceding 30 days.

Data are from the 2008 Behavioral Risk Factor Surveillance System.

Age-adjusted percentage of coronary heart disease among adults.

Data are from the 2010 Behavioral Risk Factor Surveillance System.

Figure 1.
Distribution of insufficient sleep and coronary heart disease across the United States.

Figure 2. Conceptual model of relationship among social and environmental factors, sleep, and cardiovascular disease outcomes.