Sleep as a Potential Fundamental Contributor to Disparities in Cardiovascular Health

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Abstract

Optimal sleep is integral to health but is commonly not obtained. Despite its wide-ranging public health impact, sleep health is considered only rarely by policy makers, employers, schools, and others whose policies and structures can adversely affect sleep. An inadequate duration of sleep and poor-quality sleep are prevalent in minority and low-income populations, and may be fundamental to racial and socioeconomic status inequities that contribute to a range of health conditions, including cardiovascular disease (CVD). This review examines the relationship between sleep and disparities in CVD. We describe the public health importance of sleep and the role of sleep duration, as well as the two most common disorders (sleep apnea and insomnia) as risk factors for a number of chronic diseases. We use a multilevel model focused on population health and health disparities, which is based on the notion that individual behaviors, such as sleep, are influenced by complex and dynamic interrelations among individuals and their physical and social environments. We also describe modifiable factors that contribute to insufficient sleep and circadian misalignment, propose potential interventions in various sectors (e.g., neighborhoods, schools, workplaces) that can address social structures that contribute to disparities, and recommend areas for future research. Integrating sleep into public health research will identify novel approaches for closing gaps in health disparities.

INTRODUCTION

The Institute of Medicine identified inadequate sleep and sleep disorders as public health issues in a 2006 report (36). This report estimated that although 50 million to 70 million Americans have a chronic sleep disorder, there is only a low awareness of sleep health, both among the general public and in professional communities. It also highlighted the high prevalence of sleep apnea and short sleep duration in Blacks, as well as the potential for these problems to contribute to chronic health conditions. The biological, social, or environmental bases of these sleep disorders and associated health disparities were not addressed. This review aims to further examine the relationship between sleep and disparities in relevant chronic diseases, with an emphasis on cardiovascular disease (CVD). Health disparities or inequities are defined as differences in health occurring between groups (such as those defined by race or ethnicity, or socioeconomic status) that are not only unnecessary and avoidable, but are also unfair and unjust (20). Disparities in health associated with race or ethnicity and socioeconomic status (SES) are embedded in larger historical, geographical, sociocultural, economic, as well as political contexts (163). Inequality in the built and social environments underlies key health disparities and prevalent established risk factors for CVD (e.g., physical inactivity, obesity) (57). Although an inadequate duration of sleep and poor sleep quality may substantially contribute to inequities associated with race and SES for a wide range of health conditions, sleep health is understudied by researchers and underappreciated by the general public, policy makers, and other stakeholders.

We begin with an overview of the physiology of sleep and the mechanisms by which sleep may increase the risk of chronic diseases for which persistent disparities have been identified by race and ethnicity as well as SES. We emphasize CVD as a particularly underappreciated potential consequence of suboptimal sleep, and focus on sleep duration as well as the two most common sleep disorders, obstructive sleep apnea (OSA) and insomnia. We then describe disparities associated with race or ethnicity and SES in sleep and sleep disorders, focusing on disparities in sleep among Blacks and Whites as data for other races and ethnicities are sparse. We subsequently present a conceptual framework for how the environmental context likely affects racial, ethnic, and socioeconomic sleep-related disparities in health. We conclude by suggesting critical areas for future research that will help to unpack the complex interplay among sleep, health, and health disparities, and will provide targets for novel sleep-focused interventions that may reduce persistent disparities in CVD. We suggest that understanding the complex interplay among sleep, social determinants of health, and cardiovascular health is critical if we are to design, implement, and evaluate clinical and public health initiatives for improving overall population health in addition to the health of racial and ethnic minorities as well as low-SES populations.

THE PUBLIC HEALTH IMPORTANCE OF SLEEP

Overview of Sleep Physiology

Sleep is an essential neurophysiological state that is an integral part of overall health and a source of physiological and psychological resilience (155). Universally across species, sleep is considered critical for rest and the restoration of brain and body functions. Sleep also is important for learning and memory consolidation. Major physiological functions influenced by sleep include protein synthesis, the release of hormones, and modulation of the autonomic nervous system.

There are distinct stages of sleep, characterized by different patterns of brain electroencephalographic activity. Typically, sleep begins in a light stage (termed stage N1), and during repetitive cycles lasting approximately 90 minutes, it progresses into deeper periods of non-rapid eye

movement (non-REM) sleep (stages N2 and N3) and then to REM sleep. Stage N3 (also termed slow-wave sleep) is when the brain is least likely to be aroused by external stimuli and when growth hormone and other hormones important for metabolism are released. Autonomic nervous system activity—which is critical in regulating cardiovascular functions—varies with sleep stage and with parasympathetic nervous system tone—being highest during N3 sleep—and sympathetic activation—which is highest during REM sleep. Regular transition through the stages of sleep (without excessive arousals or fragmentation) is needed for sleep to be restorative. The curtailment of overall sleep duration, disruption of the normal cycling of sleep stages, the selective curtailment of deeper sleep, increased sleep fragmentation, and abnormalities in breathing associated with sleep each can lead to acute and chronic health problems and contribute to neuroendocrine abnormalities that affect, for instance, lipid and glucose metabolism as well as vascular health. Among the physiological pathways influenced by sleep are the hypothalamic-pituitary-adrenal axis, the autonomic nervous system, the release of proinflammatory hormones, glucose homeostasis, and vascular control (23, 117).

During a person's life, sleep duration decreases from average values of approximately 16 hours a day in infancy to 7 or 8 hours in adulthood. At any given age, there is likely some interindividual variability in the duration of sleep that is associated with optimal health and functioning. Most studies suggest that shorter durations of sleep—consisting of fewer than 11 hours per night in infancy, fewer than 7 hours per night in adolescence, and fewer than 6 hours per night in adulthood—are associated with adverse health outcomes.

Sleep Disorders and Public Health

The two most common sleep disorders are OSA and insomnia, which each affect approximately 15% of the population (121, 123). OSA is the occurrence of repetitive periods of obstructed breathing during sleep (apneas and hypopneas), associated with drops in oxygen levels, arousals, and mechanical stresses on the heart and lungs. It is associated with symptoms of sleep disruption, snoring, and daytime sleepiness. Insomnia is a disorder characterized by chronic difficulties in initiating or maintaining sleep, or frequent early morning awakenings. In addition, sleep may be impaired due to irregular sleep patterns, particularly when sleep occurs outside of the normal sleep-wake circadian cycles, as occurs in shift workers and which is referred to as circadian misalignment (45, 131).

Sleep deficiency is defined as an insufficient quantity or inadequate quality of sleep relative to that needed for optimal health, performance, and well-being (24, 30). Although it is unclear whether sleep duration has decreased over time (12), it is estimated that 50 million to 70 million Americans suffer from a chronic sleep disorder. For instance, more than 12 million Americans have OSA (37). Regarding performance, suboptimal sleep contributes to poor performance in everyday activities, including academic underachievement and behavioral problems in children and adolescents, in addition to problems with work-related productivity among adults (36). Inadequate sleep is also associated with an increased risk of motor vehicle crashes and occupational injuries (34, 64, 103, 131). Almost 20% of all serious car-crash injuries in the general population are associated with driver sleepiness.

The recent recognition of the population impact of sleep deficiency has informed the Healthy People 2020 objectives for sleep health (36), which include (*a*) increasing the proportion of adolescents obtaining adequate sleep (baseline, 31%), (*b*) increasing the proportion of adults obtaining adequate sleep (baseline, 63%), (*c*) decreasing the number of motor-vehicle incidents attributed to drowsy driving (baseline, 2.7/100 million miles), and (*d*) increasing the proportion of adults with apnea symptoms who seek medical attention (baseline, 10%).

Sleep Disorders and Risk of Chronic Disease

Billions of dollars each year are spent on direct medical costs related to sleep disorders (160). Suboptimal sleep is associated with mood disorders and poorer physical health outcomes, including an increased incidence, progression, and severity of CVD, diabetes, obesity, cancer, and premature mortality (10, 24, 53, 54, 118). This section briefly reviews the evidence linking sleep with chronic-disease outcomes, and is followed by a more detailed section examining CVD.

Weight gain and obesity. Findings in 31 cross-sectional and 5 prospective cohort studies of children suggested that short duration of sleep was strongly and consistently associated with concurrent and future obesity (124). A systematic review of 30 studies among children and adults found that self-reported short sleepers were 55% more likely to be obese than those reporting at least 7 hours of sleep per night (30).

Hypertension. Epidemiological studies have shown statistically significant, independent associations between habitual sleep duration (especially during middle age) and OSA, insomnia, restless leg syndrome, as well as periodic limb movement, and an increased risk or prevalence of hypertension (26). Approximately 50% of patients with OSA are hypertensive, and more than 30% of patients with hypertension have OSA (165). OSA is also present in up to 90% of patients with resistant hypertension (165), which is more commonly observed in Blacks. Blood pressure normally decreases by approximately 10% during sleep compared with wakefulness, a pattern termed dipping. Non-dipping blood pressure, associated with an increased risk of CVD and mortality (44), is particularly common in Blacks, and can occur secondary to OSA (159), secondary to intermittent hypoxia, and due to arousals from sleep (18). Activation of the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system as seen in insomnia may also increase the risk of hypertension (15).

Diabetes. Both the quantity and quality of sleep have been demonstrated to significantly predict the risk of type 2 diabetes (29). Snoring and OSA also have been associated with abnormal glucose control and risk of diabetes, with some evidence that treatment of OSA improves this risk (150). Insomnia has been associated with a relative risk ranging from 1.84 to 2.95 (157).

Cancer. The duration of sleep depends on the circadian rhythm that controls a variety of key cellular functions, and disruption of this rhythm has been implicated in the risk of cancer. Both short durations and long durations of sleep have been associated with an increase in the risk of colorectal cancer in postmenopausal women, and short sleep has been associated with breast cancer (158). OSA also has been associated with cancer incidence (27) and mortality (119), and attributed to hypoxemia that influences angiogenesis, apoptosis, and tumor metastases.

Mood disorders. Disturbances in sleep and circadian rhythm are common in many mood and psychiatric disorders (10). There is evidence that these associations are bidirectional, and some may represent abnormalities in common neuropsychiatric pathways. Sleep disturbances often precede the onset of anxiety and depression, and are risk factors for suicidality and relapse in depression (25).

Behavior and cognition. Insufficient sleep and OSA have been linked to externalizing behaviors (negative behaviors that are directed toward the external environment, such as impulsivity, fighting, and refusal to follow rules or laws), emotion regulation, internalizing behaviors (negative

behaviors that are directed towards oneself, such as social withdrawal), and executive functioning (6, 35). Untreated OSA has been linked to poor performance in school (58), and appropriate recognition and surgical treatment of OSA in children with attention deficit hyperactivity disorder may prevent the need for long-term treatment with stimulants (89). Adults with OSA have also been shown to have higher rates of divorce (66). Thus, sleep disturbances adversely affect behaviors and physiological processes that are critical for social and cognitive development, as well as for academic and occupational performance (56, 58, 149).

Mortality. Given the number of chronic health conditions associated with suboptimal sleep, it is not surprising that it is also associated with increased mortality (53). Among 16 studies, the pooled relative risk (RR) for all-cause mortality for short sleep duration was 1.10 [95% confidence interval (CI), 1.06–1.15] (53). Similarly, among the 17 studies reporting on long sleep duration and mortality, the pooled RRs comparing the long sleepers with medium-length sleepers were 1.23 (95% CI, 1.17–1.30) for all-cause mortality, 1.38 (95% CI, 1.13–1.69) for cardiovascular-related mortality, and 1.21 (95% CI, 1.11–1.32) for cancer-related mortality (53).

THE ROLE OF SLEEP IN DISPARITIES IN CARDIOVASCULAR DISEASE

We briefly review disparities in CVD prior to describing the relationship between sleep and CVD. We provide an overview of the evidence for disparities in sleep and sleep disorders, and then introduce a conceptual framework to illustrate the potential relationship between sleep and disparities in CVD from a multilevel perspective.

Disparities in Cardiovascular Disease

Despite advances in preventing CVD, certain racial and ethnic groups as well as low-SES groups continue to have a disproportionately high prevalence of CVD (42, 94, 111, 137). **Table 1** shows the age-adjusted death rates and potential years of life lost for all causes, heart disease, and cerebrovascular disease by race, in addition to the prevalence of heart disease by SES both between and within racial and ethnic populations. Disparities in CVD are attributable to an increased prevalence of risk factors, which often have early onset and are poorly controlled. Hypertension that is difficult to control is considered a lynchpin that contributes to excessive rates of heart failure and stroke among racial and ethnic minorities (especially Blacks) (17, 52, 110). Obesity, abnormal glucose metabolism, and frank type 2 diabetes also are more common in most populations of racial and ethnic minorities (19, 41, 86, 120).

Sleep and Risk of Cardiovascular Disease

Some health outcomes most notably associated with CVD (e.g., obesity, hypertension, and diabetes) are also affected by suboptimal sleep. Sleep abnormalities are linked to abnormalities in blood pressure, lipid and glucose metabolism, and weight, and thus may significantly contribute to excess CVD. Intermediate pathways by which sleep influences CVD include its effects on diurnal patterns of blood pressure and heart rate, insulin sensitivity, the activity of the autonomic nervous system, and salt and fluid homeostasis (9, 67, 96). The intermittent hypoxemia and swings in intrathoracic pressure that occur with OSA also negatively impact cardiovascular health through their adverse effects on endothelial function and myocardial contractility, as well as the induction of oxidative stress and systemic inflammation (18). Sleep also may indirectly influence CVD risk via its effects on behaviors such as diet and physical activity (116). For instance, sleep deprivation

Table 1 Age-adjusted death rates and potential years of life lost for all causes, heart disease, and cerebrovascular disease by race, and by prevalence of heart disease and socioeconomic status between and within racial and ethnic populations[a]

Age-adjusted death rate (per 100,000), 2010 (154)							
Cause of death	**Black**			**White**			
All causes	898.2			741.8			
Heart disease	224.9			176.9			
Cerebrovascular disease	53.0			37.7			
Years of potential life lost before age 75 years (per 100,000), 2010 (154)							
Cause of death	**Black**			**White**			
All causes	9,832.5			6,342.8			
Heart disease	1,691.1			900.9			
Cerebrovascular disease	358.1			142.7			
Relative risk by education and income within racial or ethnic population (5)							
	Relative risk compared with those having 12 years of education			**Relative risk compared with those having income $20,000–29,999**			
	≤8 years	**9–11 years**	**13+ years**	**<$10,000**	**$10,000–19,000**	**≥$30,000**	**Foreign**
Studies of heart-disease prevalence							
Longitudinal Study of Aging							
White (N = 7,003)	1.17*	1.20*	0.83	1.26*	0.98	0.92	
Black (N = 888)	1.40	0.81	1.91	1.01	1.28	1.34	
Hispanic (N = 315)	1.15	0.81	0.90	1.48	0.91	1.17	1.03
Action for Health in Diabetes AHEAD 70+							
White (N = 5,896)	1.33*	1.23*	1.00	1.31*	1.19*	1.03	
Black (N = 1,023)	2.26*	1.90*	0.85	0.78	0.94	1.09	
Hispanic (N = 406)	1.09	1.36	1.08	2.69	1.63	2.22	1.35
Health and Retirement Study HRS 51-61							
White (N = 5,936)	1.21	1.39*	0.89	1.49*	1.12	0.81*	
Black (N = 1,483)	0.82	0.78	0.75	2.21*	1.04	0.80	
Hispanic (N = 772)	1.39	1.31	1.03	2.41	1.77	1.18	1.16

Relative risk by race or ethnicity, education, and income (5)									
	Relative to non-Hispanic Whites			Relative to 12 years of education			Relative to income $20,000–29,999		
	Blacks	U.S.-born Hispanics	Foreign-born Hispanics	≤8 years	9–11 years	13+ years	<$10,000	$10,000–$19,999	≥$30,000
Studies of heart-disease prevalence									
Longitudinal Study of Aging 70+									
$N = 8{,}333$[b]	0.81*	0.88	0.91						
$N = 8{,}206$[c]	0.74*	0.82	0.82	1.24	1.18	0.85			
$N = 8{,}206$[d]	0.71*	0.81	0.86	1.19*	1.15	0.86	1.24*	1.00	0.93
Action for Health in Diabetes AHEAD 70+									
$N = 7{,}342$[b]	0.76*	0.75	0.58*						
$N = 7{,}342$[c]	0.67*	0.63*	0.49*	1.43*	1.29*	0.98			
$N = 7{,}342$[d]	0.66*	0.62*	0.48*	1.39*	1.27*	1.00	1.26*	1.17	1.04
Health and Retirement Study HRS 51-61									
$N = 9{,}456$[b]	1.16	0.67	0.71						
$N = 9{,}456$[c]	1.06	0.59*	0.60*	1.32	1.35*	0.85*			
$N = 9{,}456$[d]	0.95	0.55*	0.56*	1.13	1.24*	0.89	1.72*	1.23	0.83*

[a]Data are from References 154 and 5.

[b]Equation 1: relative risk adjusted for age and sex without controlling for socioeconomic status.

[c]Equation 2: relative risk adjusted for age, sex, and four categories of education.

[d]Equation 3: relative risk adjusted for age, sex, four categories of education, and income.

*Significant at 0.05 level or below.

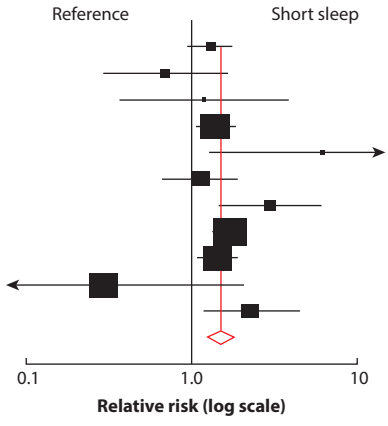
alters appetite-regulating hormones such as ghrelin and leptin, increasing hunger and thus leading to increased caloric intake (142, 168). Imaging studies indicate that sleep deprivation also affects the brain centers associated with reward behaviors, thus contributing to increased energy intake (142, 168). Sleep restriction may lead to fatigue, and result in lower levels of physical activity (168). Long sleep (lasting more than 9 hours per night) has also been linked to adverse physiological functions, and the potential mechanisms underlying the association between long sleep duration and CVD, as well as other disease risks, are not well understood (61, 87). Depression, unemployment, physical inactivity, poor health status, and chronic health conditions have been considered important contributors to suboptimal long sleep and its association with disease risk.

A meta-analysis of 15 studies found that short duration of sleep was associated with a greater risk of developing or dying of coronary heart disease (CHD) (RR, 1.48; 95% CI 1.22–1.80; $p < 0.0001$) and stroke (RR, 1.15; 95% CI, 1.00–1.31; $p = 0.047$) (**Figure 1**) (28). A long duration of

a

First author	Year	Country	Sample size	Total CHD events	Follow-up (years)	Relative risk (95% CI)
Qureshi	1997	USA	7,844	413	10.0	1.30 (0.97–1.74)
Mallon (men)	2002	Sweden	906	71	12.0	0.70 (0.29–1.65)
Mallon (women)	2002	Sweden	964	20	12.0	1.20 (0.37–3.88)
Ayas	2003	USA	71,617	934	10.0	1.39 (1.06–1.83)
Amagai (men)	2004	Japan	4,419	26	8.2	6.17 (1.29–29.6)
Meisinger (men)	2007	Germany	3,508	295	10.1	1.13 (0.66–1.91)
Meisinger (women)	2007	Germany	3,388	85	10.1	2.97 (1.47–6.02)
Shankar (men)	2008	Singapore	25,552	846	7–13	1.70 (1.34–2.15)
Shankar (women)	2008	Singapore	32,492	570	7–13	1.43 (1.09–1.89)
Ikehara (men)	2009	Japan	41,489	508	14.3	0.29 (0.04–2.05)
Ikehara (women)	2009	Japan	57,145	373	14.3	2.32 (1.19–4.51)
Combined effect: $P < 0.0001$			249,324	4,141		1.48 (1.22–1.80)

Heterogeneity: $I^2 = 44\%$; $P = 0.059$
Egger's test: $P = 0.95$



b

First author	Year	Country	Sample size	Total CHD events	Follow-up (years)	Relative risk (95% CI)
Qureshi	1997	USA	7,844	413	10.0	1.11 (0.81–1.51)
Mallon (men)	2002	Sweden	906	71	25.0	2.20 (1.05–4.64)
Mallon (women)	2002	Sweden	964	20	25.0	0.70 (0.101–5.05)
Ayas	2003	USA	71,617	934	10.0	1.36 (1.021–0.83)
Amagai (men)	2004	Japan	4,419	26	8.2	1.30 (0.381–4.37)
Amagai (women)	2004	Japan	6,906	28	8.2	15.96 (2.001–127.4)
Meisinger (men)	2007	Germany	3,508	295	10.1	1.07 (0.751–1.53)
Meisinger (women)	2007	Germany	3,388	85	10.1	1.40 (0.751–2.63)
Shankar (men)	2008	Singapore	25,552	846	7–13	1.88 (1.481–2.38)
Shankar (women)	2008	Singapore	32,492	570	7–13	1.67 (1.241–2.23)
Ikehara (men)	2009	Japan	41,489	508	14.3	1.12 (0.771–1.62)
Ikehara (women)	2009	Japan	57,145	373	14.3	1.04 (0.631–1.73)
Combined effect: $P = 0.0005$			256,230	4,169		1.38 (1.15–1.66)

Heterogeneity: $I^2 = 49\%$; $P = 0.028$
Egger's test: $P = 0.92$

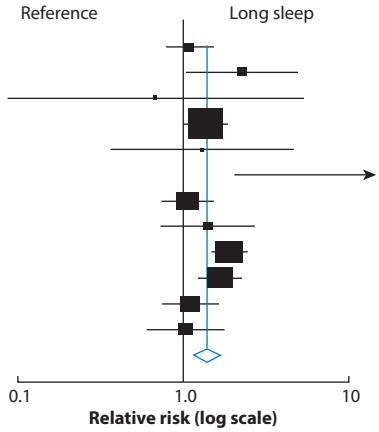


Figure 1

Forest plots of the risk of developing or dying of coronary heart disease associated with (*a*) short duration of sleep compared with the reference group and (*b*) long duration of sleep compared with the reference group (from Reference 28 by permission of Oxford University Press). Abbreviations, CHD, coronary heart disease; CI, confidence interval.

sleep was also associated with a greater risk of CHD (RR, 1.38; 95% CI, 1.15–1.66; $p = 0.0005$), stroke (RR, 1.65; 95% CI, 1.45–1.87; $p < 0.0001$), and total CVD (RR, 1.41; 95% CI, 1.19–1.68; $p < 0.0001$) (28). Epidemiological studies have also established OSA as a risk factor for incident hypertension (105), heart failure, CHD, and stroke (129, 148). Moderate to severe OSA doubles the risk for stroke, and increases the risk for CHD by 30%. Observational studies suggest that treating OSA reduces CVD and CVD-related mortality (106).

Socioeconomic Disparities in Sleep and Sleep Disorders

Emerging data indicate that individuals from disadvantaged neighborhoods and of low SES experience high rates of extreme variations in durations of sleep, poor sleep quality, as well as OSA and insomnia (55, 71, 104, 112). Research investigating the basis for disparities in sleep is in its early stages, but evidence suggests that greater exposure to stressors (e.g., neighborhood, occupational, psychosocial) and environmental exposures to tobacco, allergens, and pollutants may adversely influence the quality and duration of sleep and also exacerbate OSA (72, 77, 83). Influenced by SES, acculturation, which involves acquiring the cultural elements of the dominant society (e.g., food choices, language, music), may also negatively impact sleep quality and increase the risk for sleep disorders. Alcohol consumption, depression, shift work, unemployment, physical inactivity, and chronic health conditions also adversely influence sleep (65, 134, 141). Furthermore, despite the higher prevalence of sleep disturbances in low SES groups, these disorders are often more underrecognized and undertreated in these groups.

Racial and Ethnic Disparities in Sleep and Sleep Disorders

The following section describes racial and ethnic disparities in sleep duration, obstructive sleep apnea, and insomnia.

Sleep duration. Compared with Whites, Blacks are nearly twice as likely to report short durations of sleep (31%) (92, 146). A meta-analysis of 14 studies found larger effect sizes in studies using objective measures compared with self-reported measures of sleep duration (132). Blacks are also more than 60% more likely to report a long duration of sleep (14% report a long duration of sleep) (68). Less research has examined sleep health in Hispanics, but evidence suggests that Hispanics (or this group), too, may have increased risks for both a short and long duration of sleep compared with non-Hispanic Whites (68, 92, 101) when confounders have been controlled. Blacks may also be at risk for more severe consequences from extreme durations of sleep than Whites. Analysis of data from the National Health Interview Survey has revealed that, among individuals reporting short or long duration of sleep, Blacks were at greater risk of diabetes than were Whites (169), even when controlling for age, sex, and income. More work is needed to investigate whether similar effects are present among diverse populations living in the same built and social environments, among other racial and ethnic minority groups, and for a range of CVD outcomes. However, extreme durations of sleep appear to be disproportionately common in low-income groups and in minority racial and ethnic groups, and are associated with intermediate CVD mechanisms as well as subclinical and clinical CVD. The limited longitudinal data available suggest that an extreme duration of sleep may mediate a portion of the increased burden of CVD observed in Blacks and Hispanics.

Obstructive sleep apnea. White, Black, and Hispanic Americans (19–20%) are about twice as likely as Asian Americans (10%) to have been diagnosed with a sleep disorder, including OSA (1). The prevalence of OSA is more than twice as high in Blacks (14%) than in Whites (6%) or

Asians (4%) (33), and it is 4–6 times higher in Black children (6–8%) than in White children (16). A cross-sectional study of 280 patients with OSA found that relative to Whites (*a*) Blacks were significantly more obese and had higher rates of hypertension at the time they were diagnosed with sleep apnea; (*b*) Black females were diagnosed at a significantly younger age than were White females; and (*c*) Black males had a significantly lower oxygen saturation level than did White males (109). Thus, Blacks may experience earlier and more severe presentations of OSA than Whites, and OSA may contribute to this population's lifelong increased risk of CVD.

Insomnia. Racial differences in insomnia are not well understood (68). Insomnia is diagnosed more often in Whites (10%) than in Asians (4%) and Blacks (3%). In surveys (32) and diary (130) studies, Whites report more trouble falling asleep and staying asleep than Blacks and Hispanics (32). In contrast, cross-sectional (14), survey (125), and prospective studies (138) have found that non-Whites suffer from chronic insomnia more often, even when controlling for potential confounders and insomnia symptoms at baseline. The 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., the use of validated self-report measures or prospective designs) found evidence of racial differences in insomnia.

A CONCEPTUAL FRAMEWORK FOR THE ROLE OF SLEEP IN DISPARITIES IN CARDIOVASCULAR DISEASE

As previously described, sleep impacts a number of physiological factors that influence CVD outcomes (3). Socially determined factors and exposures (e.g., occupational stressors, discrimination, and access and adherence to treatment) can influence the same mechanisms (65, 84, 139). Thus, we examine in more detail the socially mediated pathways by which sleep deficiency may be driving disparities in CVD.

We draw upon a model developed by the National Cancer Institute's Centers on Population Health and Health Disparities (**Figure 2**) (161). The model is based on the notion that individual behaviors, such as sleep, are influenced by complex and dynamic interrelations among individuals and their physical, social, and institutional environments throughout their life. This model provides a basis for understanding the complex array of factors likely to influence sleep, and the role of sleep in health disparities. Biological pathways and responses are a key part of this model, as illustrated below. Here we focus on individual, physical, social, and institutional factors.

Distal determinants, as described in the figure, are considered fundamental causes of population health and disparities in health because their influence contributes to variations in health and disease. An example of a distal determinant is the establishment of city ordinances that control the working hours of construction sites to protect residents from noise that could disrupt sleep. Intermediate determinants, such as neighborhoods, represent the physical and social contexts where distal determinants are realized. The availability and accessibility of certain tangible and social resources (e.g., social cohesion, lighting) may influence characteristics of the sleep environment. It is believed that intermediate determinants link the environment to individuals, and influence biological responses and more proximal determinants (e.g., stress or anxiety, sleep homeostasis, the misalignment of circadian rhythm).

Proximal Factors: Individual Risk Behaviors

Lifestyle factors, including poor nutrition, a lack of physical activity, alcohol consumption, and smoking, are established risk factors for CVD and can influence sleep quality. Sleep behaviors,

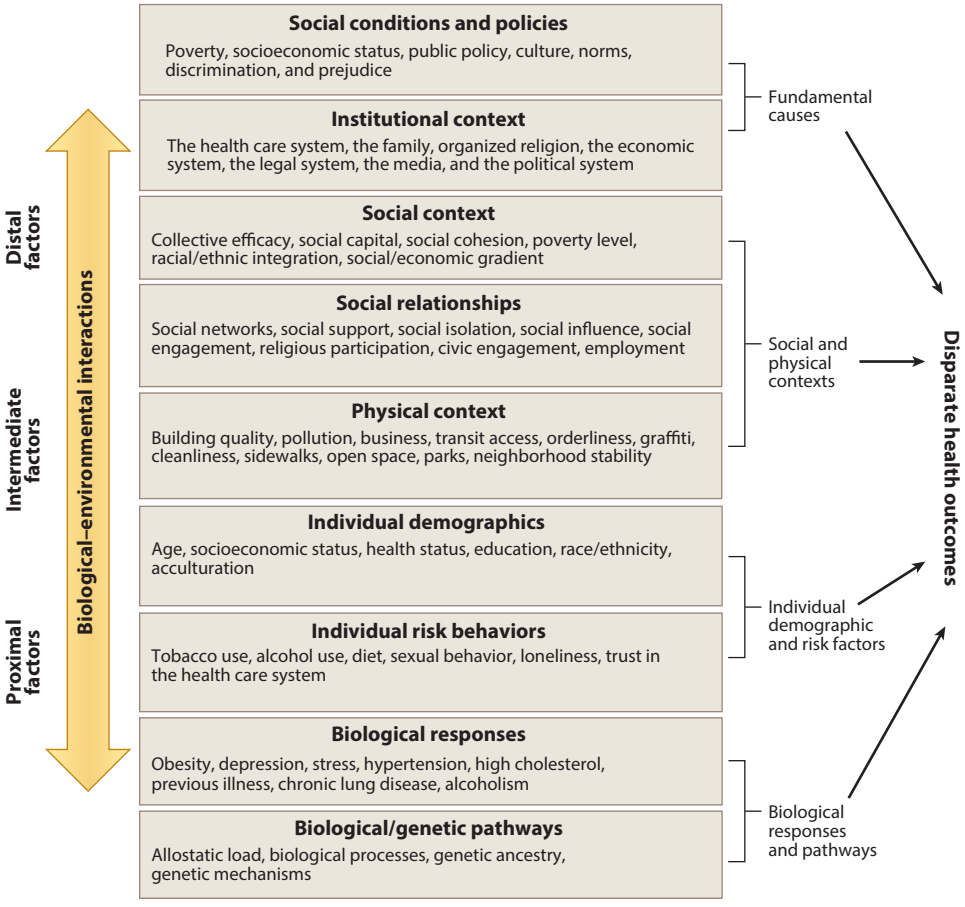


Figure 2

The socioecological model and determinants of sleep and cardiovascular health. Adapted with permission from Reference 161. Abbreviation: CVD, cardiovascular disease.

such as having a regular bedtime and limiting the use of substances (such as alcohol and tobacco) and certain activities (such as watching TV in bed), which are considered sleep hygiene, also influence the quality and duration of sleep (7, 60, 130).

Recent trends in the use of technology and its affordability have resulted in a 24-hour society where individuals are perpetually available and capable of communicating and engaging in work in ways that may displace sleep differentially across groups (39, 62). Screen exposure is particularly disruptive to sleep due to the alerting effects of light (especially blue light) on the sleep centers in the brain. According to a 2011 national poll, 90% of Americans reported using a technological device in the hour before going to bed, and young adults went to bed significantly later than other age groups on both weekdays and weekend nights (59). Unlike with passive technological devices (e.g., TV), the more interactive the technological devices (i.e., computers, cell phones, video games) that are used in the hour before bed, the more likely it is that the user will have difficulties falling asleep and will have unrefreshing sleep. Although Blacks use the Internet less than Whites (87% of Whites and 80% of Blacks are Internet users), Blacks and Whites have similar rates of using social media, especially on mobile platforms; 73% of Black Internet users—and 96%

of those ages 18–29—use a social networking site of some kind (51). The closing of the digital divide in technology may inadvertently contribute to disparities in health.

Proximal Factors: Individual Demographics

The following section describes proximal factors like acculturation and other demographic factors that may have a notable influence on sleep health and sleep disorders.

Acculturation. Recent evidence suggests that the harmful health effects of acculturation, which involves the acquisition of the cultural elements of mainstream society (e.g., in choosing food, language, and music), extend to sleep duration among Hispanics (38, 78, 97). A nationally representative study found that Mexican Americans were 44% more likely to report a short duration of sleep than were Mexican immigrants when confounders were controlled (72). This effect was modestly attenuated after considering the effects of smoking and self-reported stress. A study of more than 300 women of Mexican descent found that acculturation (measured by language preference and socialization in the United States before the age of 18 years) predicted self-reported sleep disturbance (75). These studies provide initial evidence highlighting acculturation as a mechanism that predisposes Mexican Americans to higher levels of sleep deficiency than Whites.

Other demographic factors. Demographic factors are important to consider, given their associations with sleep disorders. In particular, OSA increases in prevalence and severity with advancing age and is more severe in men than women, likely due to the influence of sex hormones on airway patency and ventilation (4, 128). OSA has also been identified to be a risk factor for divorce (66), and in women the risk of OSA is markedly increased after menopause (13). Furthermore, insomnia has been shown to be more common in women than men (167).

Intermediate Factors: Physical Context—Built Environments (Neighborhood and Housing Disadvantages)

Highlighting the interplay across the levels of influence, population health, insufficient sleep, and OSA are associated with individual-level factors, such as sleep hygiene and obesity, and also with neighborhood disadvantage and low SES (71, 77). Members of racial and ethnic minority groups are more likely than Whites to live in disadvantaged neighborhoods (145, 163), and the adverse effects of stressors and exposures in these neighborhoods may contribute to disparities in CVD via an influence on sleep. Residents living in poorer neighborhoods are more likely to be exposed to factors that may contribute to sleep deficiency, such as inopportune exposure to light, noise, allergens, and irritants—e.g., environmental tobacco (95, 107) or air pollution (48, 49). Some of these factors (such as particulate air pollution) may also contribute to abnormalities in the autonomic nervous system and increase cardiovascular morbidity. Neighborhood disorder may also increase the prevalence of both poor self-reported sleep quality (71, 77) and sleep-disordered breathing (166).

Intermediate Factors: Social Relationships

In the United States, sleep patterns and sleep disorders vary by social factor (e.g., discriminatory practices), and tend to be tied to sociodemographic factors (e.g., neighborhood characteristics) that are likely to contribute early in life to disparities in educational attainment, economic opportunity and productivity, and health.

Family influences. The sleep of caregivers influences the sleep of children, and vice versa. Sleep behaviors also reflect the influences of interrelated social, cultural, and environmental factors operating within households. For example, children's sleep may be impacted by factors such as family routines, parenting styles (e.g., whether there are sleep routines and curfews, or TVs in bedrooms), family illnesses or accidents, depressed parental mood, parents' work schedules, as well as exposure to intimate partner violence and other traumatic life events. In fact, adverse childhood experiences have been associated with self-reported sleep disturbances in adulthood (32).

A highly important but understudied area is the role that stress may have in affecting disparities in sleep and CVD. Stress is linked to both sleep disturbances and CVD; thus, it is critical to carefully consider the interrelationships among stress, sleep, and CVD as fundamental contributors to health disparities. This is supported by (*a*) the mediating role of stress in linking socioeconomic disadvantage and risk of CVD (2, 43); (*b*) physiological links among stress, arousal, and disrupted sleep (88, 100); (*c*) associations among negative emotions and lifelong discrimination with impaired sleep (147); and (*d*) the increase in CVD risk factors seen in individuals with impaired sleep (8, 87). Prior mediation analyses have estimated that impaired sleep may explain 10–25% of the variance in health outcomes associated with low SES (70). However, the mediation analyses were conducted using cross-sectional data, and did not objectively measure CVD risk, thus precluding definitive assessment. The availability of social support to overcome stressors that impact sleep is also important. Although not well studied, sleep is also likely to be influenced by psychosocial interactions within households—such as chaotic family routines, mother-child stress, the level of autonomy allowed and practiced by each family member, and poor sleep patterns of family members (69, 70, 144)—all of which may influence stress responses leading to increased arousal, a known mediator of disturbed sleep (122, 143).

Intermediate Factors: Social Context

The following section discusses racial discrimination on multiple levels (e.g., institutional, personally mediated, and internalized) as an example of an intermediate or contextual factor that may influence disparities in sleep health.

Racial discrimination. Racial discrimination may be an important determinant of racial disparities in health. For instance, a study of racially salient chronic stress—known as racism-related vigilance—and sleep difficulty found that Blacks reported greater levels of racism-related vigilance, and greater levels of sleep difficulty compared with Whites (76). Institutional and interpersonal racial discrimination may lead to chronic psychosocial stress among racial and ethnic minorities (108, 164). Both objective and perceived racial discrimination are psychosocial stressors that are disproportionately experienced by racial and ethnic minorities, and they may be implicated in sleep disparities (31, 63, 76, 98, 139, 152, 162). Reports of perceived discrimination were positively associated with an increased risk of sleep disturbance and daytime fatigue in a survey of more than 7,000 Black and White adults (63), and there were similar findings in a smaller study of Hispanics (147). Perceived discrimination has also been shown to be associated with reduced time in deeper sleep (stage N3) (151, 156). Hispanics showed a smaller but nonsignificant association, and Whites showed no association between racial vigilance and sleep difficulty (76).

Another study found that multiple levels of racism, including interpersonal experiences of racial discrimination and the internalization of negative racial bias, operate jointly to accelerate vascular aging among Black men as measured by telomere length and which involves endothelial cells, smooth muscle cells, and cardiomyocytes (31, 133). The household ratio of income-to-poverty threshold and the interaction between racial discrimination and implicit racial bias were

significantly associated with leukocyte telomere length (31). More work is needed to unpack the effects of discrimination on sleep, and the effect of microaggressions or everyday subtle indignities should also be studied. Nonetheless, evidence suggests that the stress of discrimination influences the quality and duration of sleep among racial and ethnic minority groups and, thus, may have important downstream effects on CVD outcomes.

Distal Factors: Institutional Context

The following section describes distal or institutional factors (e.g., occupational characteristics) that are likely to directly or indirectly influence sleep and sleep disparities.

Occupational patterns. There is considerable epidemiological evidence that shift work is associated with a range of problems including lost work-related productivity, poor concentration, absenteeism, accidents, errors, injuries, and fatalities, as well as elevated blood pressure, obesity, cancer, and diabetes (46, 74, 85, 88, 91, 99, 114, 115, 136). Shift work is more common in Blacks than Whites (100, 127), and is likely an important contributor to racial differences in short durations of sleep (21).

Short duration of sleep prevalence varies by industry and occupation among US workers (102), and work has been shown to affect sleep through the requirement for long or extended work hours, rotating or night-shift work, and job-related stress (93, 126, 153). US Blacks may be at particularly high risk for the adverse influences of sleep deficiency on morbidity and mortality (78, 90). Blacks, compared with Whites, are more likely to report job-related stress, to work in low control–high demand positions (especially those with low decision-making power), to work more than one low-wage job, to live in poverty despite employment, and to experience discrimination (both objective and perceived) (47, 146, 151, 152). It is also likely that workplace exposures to airborne irritants may contribute to disparities in OSA.

Using nationally representative data, racial and ethnic differences in the prevalence of short duration of sleep by industry of employment and occupation were observed (79). Blacks, regardless of occupational status, had a higher prevalence of short sleep than their White counterparts, and the disparity was widest among professionals. Additionally, the prevalence of short sleep increased among Blacks as professional responsibility increased, but among Whites it decreased as professional roles increased. The high prevalence of short durations of sleep among professional Blacks may be partly attributable to limited or less well-connected professional or social networks that can provide financial and emotional support; more taxing interpersonal relationships related to, for example, John Henryism (a coping strategy where prolonged exposure to stressors such as discrimination leads to high levels of effort that have detrimental health consequences); and discrimination (e.g., microaggressions) in the workplace (81). Discrimination may play an important part in producing psychosocial stress in addition to job strain or having limited control over job demands, as illustrated by the well-known Karasek and Theorell demand-control model that has established an association between high-demand/low-control jobs and heart disease (84, 90). The role of insufficient sleep in the context of upward social mobility deserves further study.

Treatment access and adherence. Sources of disparities in health care occur at the level of the provider (e.g., bias, clinical uncertainty, beliefs or stereotypes about the behavior or health of minority patients) and the resource level of the health care systems (e.g., lack of interpretation and translation services, time pressures on physicians, geographical availability, instability in the financing and delivery of health care services) (140). Racial or ethnic minorities with sleep disorders may be diagnosed and treated later, in part due to having reduced access to screening, diagnosis,

and interventions, and this may also potentially have an important role in disparities in CVD (16). Federally qualified health centers serve as safety nets for more than 19.5 million Americans, mostly those from disadvantaged backgrounds. Although care-paths for managing sleep disorders are evolving, diagnostic testing for sleep disorders and the management of OSA usually require the involvement of sleep-medicine specialists, who often do not practice in these settings. Thus, health disparities could be exacerbated among underserved populations that have limited access to sleep-focused specialty services in federally qualified health centers. Barriers to referrals for specialty care caused by insurance status and geographical location also may lead to persistent disparities. The limited training that primary care providers receive in sleep medicine contributes to the high proportion of patients with OSA and insomnia who are not diagnosed or treated adequately (82). Disparities in care and outcomes are likely to worsen without interventions designed to improve access.

Evidence also suggests that independent of access to treatment, adherence to sleep-disorder treatments is lower among Blacks than Whites (22, 135), which may unnecessarily increase the incidence and severity of CVD outcomes. In a clinical trial of OSA management pathways that provided standardized care to all participants, Blacks were observed to have lower treatment adherence than Whites (11). Differences in socioeconomic resources, social support, and the perceived benefits or risks of treatment may underlie racial differences in adherence (50). Although there are minimal data identifying the best targets for sleep interventions in the primary care setting, several promising areas include improving patients' sleep literacy, doctor–patient communications, care processes, organizational practices, and health care quality by incentivizing good sleep care. Lessons learned from efforts to reduce other disparities in health care may provide a useful guide.

Distal Factors: Social Conditions and Policies

There have been limited public-policy interventions designed to directly improve the population's sleep health. A growing number of school districts have implemented later start times for schools as a strategy for prolonging sleep duration for children, with some evidence that later start times lead to decreases in motor vehicle crashes among adolescents and improved school attendance (40). However, wider adoption has been limited by financial constraints in school districts that are unable to budget for altered scheduling and bus routes. Several initiatives also are under way to improve public and corporate awareness of sleep health and drowsy driving. Industries employing shift workers have begun to develop policies to mitigate the effects of lowered vigilance among workers, and medical residents are now limited in their work schedules to minimize sleep deprivation. A recent program initiated by Harvard Medical School (known as ReCharge America) aims to provide, in the workplace, companies and policy makers with knowledge, technical assistance, communications support, and tools to support healthy sleep (73). There are opportunities for the numerous federal and local government agencies that oversee public health, safety, and transportation to more actively develop policies to improve the sleep health of their employees, including those whose job responsibilities have an impact on public safety (e.g., transportation workers), and to implement policies to further mitigate drowsy driving. The Centers for Disease Control and Prevention partners with various stakeholders to conduct surveys on sleep, and national efforts are now under way to develop consensus documents on sleep needs across the lifespan. The US Department of Housing and Urban Development could play a part as those living in poorer neighborhoods are more likely to be exposed to factors that may contribute to sleep deficiency, such as inopportune exposure to light, noise, allergens, and irritants (e.g., environmental tobacco) (95, 107). Noise ordinances and a focus on establishing community links to resources to alleviate stressors that impact sleep should also be considered as useful policy strategies.

Future Directions for Research into Sleep Disparities

In this section, we describe the need for data in relation to sleep research overall, as well as in CVD and disparities in CVD, noting that there are significant methodological challenges in the extant research. Most prior sleep research relies on self-reporting, but surveys often have not been validated in the specific groups of interest. Although patient-reported outcomes are important, the inclusion of objective measures of sleep, as obtained by actigraphy or polysomnography (the gold standard), may minimize the misclassification of behaviors that may be difficult to report due to their occurrence during sleep (e.g., snoring among individuals without a bed partner or sleep latency), as well as provide quantitative data on the degree and type of sleep disruption. Actigraphy, which captures data through a small wrist-worn device, records movements over multiple days and provides reliable and valid estimates of sleep–wake periods, yielding objective measures of average and night-to-night variability in sleep duration and of sleep efficiency (a function of wake time during the sleep period) (113). Polysomnography monitors multiple physiological variables during an overnight study to identify specific sleep stages and to characterize patterns of breathing, blood oxygen levels, heart rate, and leg movements (80).

The data support the need to evaluate sleep disorders as a target for both primary and secondary reductions in CVD. To advance this research agenda, there is a need for research that examines the possible bidirectional relationships between stress and sleep, and their associations with CVD outcomes. Longitudinal or prospective studies will be critical in establishing the temporal ordering of sleep duration and sleep problems, stress, and CVD outcomes for different racial and ethnic groups. Therefore, investigators should conduct research among more diverse populations because racial and ethnic minority groups and low-income groups have been underrepresented in sleep research. Furthermore, causal modeling may help discern the interactive roles of stress and sleep on the risk of CVD. There is also a need to better measure stress across racial and ethnic groups, and to distinguish the effects of sudden, daily, and chronic stress in addition to stresses occurring during developmental periods that may be particularly relevant for their impact on sleep.

Studies are needed to identify key demographic, personality, cultural, 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 the risk of CVD in individuals of different ethnic, racial, or socioeconomic backgrounds. For example, are shift workers at higher risk for CVD if they also live in a poor, urban neighborhood?

Multilevel research could further our understanding of the influences of individual, household, and neighborhood factors on sleep, and on the relationship between sleep and CVD. Studies of environmental exposures could be enhanced by considering biological effects as measured through epigenetic studies. Given that disturbed sleep and an elevated risk of CVD emerge early in life in racial and ethnic minorities, it is crucial that these associations be evaluated in both children and adults.

In addition to focusing on risk factors, future research should also identify social, cultural, and physical factors associated with resilience or that are protective for sleep and CVD despite adverse environments. An improved understanding of the influences of acculturation on sleep may also help identify the role of a Westernized lifestyle on sleep and CVD in addition to potentially identifying how stress associated with acculturation influences sleep and CVD. The emerging data linking sleep with chronic health problems, including CVD and diabetes, provide a strong basis for the public health community to integrate sleep into investigations of behavioral risk factors, and to consider including sleep-health targets in intervention research. Achieving a sustainable population-level impact on sleep disparities will likely require coordinated efforts that link policies, systems, and environmental changes in diverse settings, such as schools, workplaces, community

centers, and residential settings (e.g., housing developments), as well as the immediate home environment. Although challenging, successful initiatives could have a high impact, given the multifaceted roles that sleep has in health and well-being.

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