Title
Sleep Health as a Determinant of Disparities in Stroke Risk and Health Outcome

Journal
Stroke, 54(2)

ISSN
0039-2499

Authors
Khot, Sandeep P
Taylor, Breana L
Longstreth, WT
et al.

Publication Date
2023-02-01

DOI
10.1161/strokeaha.122.039524

Peer reviewed

Stroke

TOPICAL REVIEW

Sleep Health as a Determinant of Disparities in Stroke Risk and Health Outcome

Sandeep P. Khot, MD, MPH; Breana L. Taylor, MD; W.T. Longstreth, Jr, MD, MPH; Arleen F. Brown, MD, PhD

ABSTRACT: Sleep is essential to human survival and overall vascular health. Sleep health encompasses the objective and subjective qualities associated with one's daily pattern of sleep and wakefulness and has become a growing clinical and public health concern. Impaired sleep duration and quality can increase stroke risk and mediate the relationship between the physical aspects of an individual's environment and disparities in stroke incidence. Here, we review observational studies evaluating the association between sleep health and cerebrovascular disease. We assess the influence on sleep of the physical environment, including the ambient environment with noise levels and the built environment. We also describe the influences on sleep health and stroke risk of social determinants of health, including the chronic stressor of racial discrimination. Finally, we discuss how changes in historical neighborhood characteristics or societal policies can influence the social factors affecting sleep health and stroke risk among socioeconomically disadvantaged groups or ethnic and racial minorities. Given the regional and racial or ethnic differences in stroke risk across the United States, an understanding of novel vascular risk factors, such as the multifaceted role of sleep health, will be critical to develop effective public policies to improve population health.

GRAPHIC ABSTRACT: A graphic abstract is available for this article.

Key Words: incidence ■ public health ■ racism ■ risk factors ■ sleep ■ social determinants of health ■ stroke

The emerging concept of "sleep health" has been underrecognized in public health, though presents a more holistic view of sleep rather than individual symptoms or disorders.[1,2] Although lacking a clear definition, sleep health is typically described as a multidimensional pattern of sleep-wakefulness that can be measured by many factors associated with sleep including: regularity, duration, continuity, efficiency, timing, daytime alertness, daytime sleepiness, satisfaction, and quality.[1,2] Formal sleep disorders[3] and other sleep disturbances may both increase the risk of vascular brain injury, both overt[4] and covert.[5] Here, we review the potential effects of poor sleep health on vascular brain injury and brain health. We also discuss factors contributing to poor sleep health, with a focus on social determinants of health. Sleep health varies with these factors, which are ripe for modifications with the aim of reducing the risk of vascular brain injury while improving brain health.

SLEEP HEALTH

Beyond its basic functional and physiologic benefits, sleep is critical for brain and vascular health. These functions include learning and memory consolidation; protein synthesis; autonomic nervous system modulation; and release of hormones, especially those important for metabolism.[6] A conceptual model of sleep health posits that the various dimensions of sleep-wake cycles can reciprocally affect and be affected by epigenetic, molecular, and cellular intermediate processes, which in turn, can affect system level processes important for health outcomes (Figure).[1] Here, we focus on social and environmental factors of sleep health but recognize other important factors such as behavioral and genetic that we will not address in this review. Research studies evaluating sleep health after stroke have mostly focused on sleep-disordered breathing, including obstructive sleep apnea, which affects an estimated 70% of stroke survivors[7] and

Correspondence to: Sandeep Khot, MD, Department of Neurology, Box 359775, Harborview Medical Center, 325 Ninth Ave, Seattle, WA 98104. Email skhot@uw.edu
This manuscript was sent to Emmanuel Touzé, Guest Editor, for review by expert referees, editorial decision, and final disposition.
Supplemental Material is available at https://www.ahajournals.org/doi/suppl/10.1161/STROKEAHA.122.039524.
For Sources of Funding and Disclosures, see page 601.

Stroke is available at www.ahajournals.org/journal/str

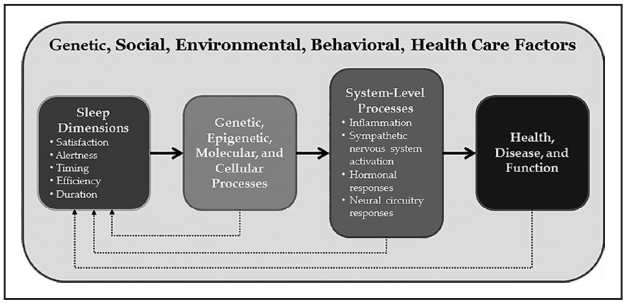

Figure. Conceptual model of sleep health.
The model posits a reciprocal relationship between sleep dimensions and various intermediate, systems-level and organism-level health outcomes. Adapted from Buysse[1] with permission. Copyright ©2014, Oxford University Press.

has been shown to be associated with both incident[8,9] and recurrent stroke.[10,11] However, sleep health can also be influenced by other factors beyond obstructive sleep apnea, including fragmented, non-restorative, delayed or insufficient sleep. Fragmented sleep can be assessed by evaluating awakenings or sleep efficiency,[12] a measure of sleep continuity and disturbed sleep, or through questionnaires evaluating sleep quality.[13] Longer or shorter habitual sleep patterns[14,15] can be associated with fragmented sleep though may also be related to underlying chronic health problems, including depression and sleep apnea. Non-restorative sleep is one of the core symptoms of insomnia and is typically defined as the subjective feeling of being unrefreshed upon awakening. It has been understudied[16,17] and is mostly measured subjectively through patient-reported outcome instruments, which have documented fatigue, tiredness, difficulty focusing, and daytime sleepiness.[17] Delayed or insufficient sleep is commonly defined as objective insomnia and measured by polysomnography,[18] though has also been examined in research studies as poor sleep with short duration as assessed by questionnaires.[19] Excessive daytime sleepiness, another important aspect of sleep health, is defined as the subjective inability to maintain attentive wakefulness and is also typically measured through patient-reported outcome instruments.

The effects of poor sleep health can lead to numerous physiologic changes related to overall vascular health, including increased insulin resistance; neuroendocrine hormone changes resulting in increased appetite; release of proinflammatory hormones; increased catecholamine secretion; and effects on growth hormone and cortisol release.[20] Many of these physiologic changes are also likely implicated in the effects of poor sleep on the development or worsening of traditional stroke risk factors, including obesity,[21] hypertension,[22] and diabetes.[23] Several studies have shown that the mechanism linking the chronic arousal in insomnia with hypertension is likely through hyperactivity of the sympathetic nervous system[24] and activation of the hypothalamic-pituitary-adrenal axis.[25] The link between chronic insomnia and diabetes risk is theorized to result from impaired glycemic control, insulin resistance, and inflammation related to chronic sleep loss.[23] Conversely, many of the more traditional stroke risk factors, including hypertension and obesity, could lead to poor sleep health.[26,27] Such reverse causation or a potential bi-directional relationship between sleep health and stroke risk factors can limit the ability to draw clear conclusions in many of the cross-sectional studies evaluating these associations.

Evidence-based guidelines recommend that adults receive 7 to 9 hours of sleep per night.[28,29] However, nearly a third of adults in the United States report short-sleep duration, defined as ≤6 hours per night, a trend which has increased in prevalence over time.[30,31] Long sleep duration, defined as ≥9 hours per night,[30] occurs in an estimated 8.5% of adults. Both short and long sleep duration have been shown to be associated with numerous adverse health outcomes, including heart disease and all-cause mortality.[32,33] Populations that are vulnerable to poor sleep health include racial and ethnic minorities and those with lower socioeconomic status (SES).[34] For example, the odds of reporting clinically significant sleep disturbance among Black adults was 3 times higher than White adults in an urban primary care setting and remained significant even after adjustment for depression, SES and disease burden (odds ratio, 2.44 [95% CI, 0.85–7.01]).[35] Similar findings have been reported among other ethnic and racial minority populations, including Mexican Americans, other Hispanics, Asians, American Indians and Alaska Natives.[16,34,36] The reasons for poor sleep health among racial and ethnic minorities have not been extensively studied, though observational cohort studies among multiple different populations provide important insights into this potentially modifiable stroke risk factor (Table).

SLEEP AND STROKE

Stroke disproportionally affects low income and ethnic and racial minority groups.[37–39] Some of these disparities in stroke incidence and outcome may be related to a higher prevalence of traditional and modifiable stroke risk factors among low-income and certain racial-ethnic groups, including smoking, hypertension, obesity, and diabetes.[40–42] However, traditional stroke risk factors may not fully account for the increased risk among low-income individuals and racial-ethnic minorities.[43–45] Sleep health may be an important, overlooked contributor to stroke disparities. Research on the association between sleep health and stroke has mostly entailed population-based, observational cohort studies. Their strengths include the prospective evaluation of multiple exposures and potential confounders, including demographic, clinical, and multiple sleep-related factors. Nonetheless their ability to address causation is limited by many potential biases,

 Stroke. 2023;54:595–604. DOI: 10.1161/STROKEAHA.122.039524

Table. Studies Evaluating Sleep Health in Relation to Stroke Risk

Study	Study type	Sample characteristics	Variables assessed	Main findings
Qureshi et al[15] NHANES (1997)	Prospective observational cohort	7844 White and Black participants	Sleep duration and daytime somnolence	Persons with both long sleep (>8 h) and daytime sleepiness almost twice as likely to suffer stroke and 50% higher mortality risk.
Chen et al[14] WHI (2008)	Prospective observational cohort	93 175 post-menopausal women	Habitual sleep patterns, defined by short (≤6 h) or long sleep duration (≥8 or ≥9 h)	Longer sleep associated with 24–70% increased stroke risk, likely independent of symptoms of sleep-disordered breathing.
Bertisch et al[18] SHHS (2018)	Prospective observational cohort	4994 cardiovascular disease-free participants	Insomnia or poor sleep and short sleep duration (<6 h) by questionnaires and at-home polysomnography	Co-occurrence of subjective insomnia and objective short sleep associated with 29% higher incident cardiovascular disease, including all stroke.
Cappuccio et al[4] (2011)	Meta-analysis	474 684 adult men and women from 8 countries	Sleep duration	Both short and long sleep associated with increased risk of coronary heart disease and stroke.

NHANES indicates National Health and Nutrition Examination Survey; SHHS, Sleep Heart Health Study; and WHI, Women's Health Initiative.

especially the possibility of residual confounding. Is the vascular injury leading to changes in sleep health, vice versa or both? Further, many observational studies of sleep quality have relied on subjective measures of sleep, which have been shown to have low association to objective sleep duration,[46,47] limiting the ability to draw accurate conclusions from these studies.

One of the earliest studies to evaluate the risk of vascular disease with sleep duration and quality was National Health and Nutrition Examination Survey (NHANES). In an analysis of sleep patterns and vascular disease among nearly 8000 Black or White adults aged 31 to 74 years and followed for 10 years, those with >12 years of education were 40% less likely to report sleeping for more than 8 hours per night (relative risk [RR]=0.6, 0.5–0.8) and 40% less likely to report excessive daytime sleepiness (RR=0.6, 0.5–0.7).[15] Black adults were also 30% more likely to report long sleep duration (RR=1.3, 1.0–1.6). The 10-year survival free of all stroke was significantly lower among those who reported both longer sleep and daytime sleepiness (88%) compared with those who reported <8 hours of sleep and no daytime sleepiness (97%). In analyses controlling for demographic and multiple stroke risk factors, both longer sleep duration and daytime somnolence were independent predictors of stroke, and participants with the combination of the 2 were nearly twice as likely as those without both conditions to suffer an incident ischemic or hemorrhagic stroke (RR=1.9, 1.2–3.1). There was no association between short sleep duration (≤6 hours) and stroke risk. In the fully adjusted model, mortality was also significantly higher in participants who reported these habitual sleep patterns and excessive daytime sleepiness (RR=1.5, 1.2–1.9) though hospitalization or death from coronary heart disease was not. The investigators postulated that the association between habitual long sleep duration and stroke risk was mediated through sleep-disordered breathing, notably a heightened physiologic need to sleep longer related to multiple awakenings from underlying sleep apnea. Limitations of NHANES include the lack of evaluation for depression, physical activity, employment status or objective sleep markers, including objective sleep duration or underlying sleep apnea. Further, causality between prolonged sleep with daytime sleepiness and increased stroke or mortality risk in this cross-sectional study cannot be ascertained, though the findings point to the possible importance of sleep health as a potentially modifiable stroke risk factor that could be screened for in certain vulnerable populations.

Some of the limitations of the NHANES were addressed in the Women's Health Initiative. Among 93 175 postmenopausal women aged 50 to 79 years followed for an average of 7.5 years, a nonlinear, *U*-shaped relationship was noted between self-reported sleep duration and ischemic stroke risk.[14] Women who reported sleep duration of 7 hours per night had the lowest risk for incident or recurrent ischemic stroke while the risk was higher in women with sleep ≤6 hours or ≥8 hours per night. Racial and ethnic minorities reported a higher percentage of short-duration sleep (19% Blacks, 14% Hispanics, 15% American Indians and 15% Asians) compared with Whites (7%), and lower SES was associated with both short and long sleep. After adjustment for demographic factors, SES, and depression, subjective short sleep (≤6 hours) had a modest association with ischemic stroke risk (RR=1.16, 1.00–1.36), but the association was no longer significant after adjustment for use of hormone therapy and relevant vascular risk factors. In contrast, subjective sleep duration ≥ 8 hours was associated with a nearly 25% increased ischemic stroke risk (RR=1.24, 1.04–1.47) and reported sleep duration ≥9 hours conferred a 70% increased risk (RR=1.70, 1.32–2.21). Women with depressive symptoms or vascular risk factors, including existing cardiovascular disease, diabetes, hypertension or hypercholesterolemia, were more likely to have both short and long sleep duration than those without these comorbid conditions. An exploratory analysis with jointly modeled effects of subjective sleep duration and frequent snoring or daytime sleepiness found that the increased stroke risk related to subjective sleep duration was likely independent of symptoms of sleep-disordered breathing. Overall, the results from Women's Health Initiative noted a modest ischemic stroke risk related to short sleep duration which

was not reported in the population of NHANES and a more robust association for long sleep duration, which was influenced though only partly explained by income, physical activity level, and depressive symptoms. The investigators postulated multiple mechanistic mediators for the increased stroke risk with long sleep duration, ranging from increased systemic inflammation with progressive atherosclerosis to circadian-related cardiac arrhythmia, though note the need for more studiers to test these hypotheses.

The findings of the Women's Health Initiative were further evaluated in the SHHS (Sleep Heart Health Study), the largest prospective cohort study with information on both subjective insomnia and at-home unattended, polysomnography-determined objective sleep markers, including sleep-disordered breathing.[18] Among 4994 participants aged >40 years and followed for a median of 11.4 years, investigators addressed the question with propensity-adjusted, logistic regression controlling for demographic factors, clinical factors, and polysomnography-evidence of sleep-disordered breathing. In a fully adjusted model, the cooccurrence of subjective insomnia and objective short sleep < 6 hours per night was associated with a 29% higher incidence of cardiovascular disease, including all stroke, compared with those with no complaints of insomnia and objective sleep for at least 6 hours per night (hazard ratio=1.29, 1.00–1.66). These results were not attenuated after adjustment for hypertension or diabetes. In the fully adjusted model, only objectively measured short sleep duration was associated with a higher mortality (hazard ratio=1.14, 1.01–1.30). The authors concluded that the unique phenotype of subjective insomnia and objective short sleep is associated with the highest incidence of major cardiovascular events, including stroke. Given the lack of such an association between short sleep and stroke risk in studies utilizing only subjective reports of short sleep, including NHANES, these findings point to the importance of considering insomnia symptoms and objective sleep duration concurrently in future prospective, observational cohort studies focused on stroke.

Sleep duration and the risk of coronary heart disease or stroke was the focus of a meta-analysis of 15 prospective, observational cohort studies of nearly 500 000 patients followed for >3 years in which sleep duration was evaluated by questionnaire.[4] Self-reported short sleep duration was associated with a near 50% increased risk of developing or dying from coronary heart disease (RR=1.48, 1.22–1.80) and a more modest 15% risk of all stroke (RR=1.15, 1.00–1.31). Long sleep duration had a more modest association for coronary heart disease than short sleep duration (RR=1.38, 1.15–1.66) but a more robust association with developing or dying from stroke (RR=1.65, 1.45–1.87). These associations suggest that sleep health should be further evaluated in future studies as a potentially modifiable risk factor for stroke.

SLEEP AND SOCIAL DETERMINANTS OF HEALTH

If sleep health is a contributor or risk factor for stroke, what are some of the factors associated with such poor sleep health? Many such factors likely exist, including genetic, environmental, and social determinants of health (Table S1). The National Cancer Institute's Centers on Population Health and Health Disparities developed a model based on the notion that individual risk behaviors, including sleep behaviors, are influenced by complex and dynamic interrelations among individuals and their physical, social, and institutional environments (Figure S1).[6,48] In this model, social conditions—such as living in an impoverished community, occupying a high-stress or demanding job, or being exposed to racial discrimination—can lead to disparate health outcomes, as can policies promoting such conditions.

Sleep and the Physical Environment

Here, we focus on 2 under-appreciated physical aspects of an individual's environment that can contribute to sleep health: the ambient and built environment.[49] The ambient environment, which encompasses noise, light and air pollution, can impact one's ability to fall and remain asleep. For example, a quiet night-time environment can promote lower stress and healthy sleep, while the opposite can result in higher stress and poor sleep due to environmental factors such as increased light exposure, traffic, and air pollution.[50] The built environment, defined as the neighborhood space around one's home, work, or school, can influence healthy behaviors like physical activity, which may improve sleep health but can also have negative impacts on sleep health if features of the environment contribute to increased stress among residents exposed to that environment (Figure S2).[49]

Many features of the built environment that allow individuals to be active in their neighborhood can also affect sleep health, such as public transport to do errands or commute to work; green space and sidewalks to promote walking; and places of social connectivity to prevent social isolation. In a cross-sectional analysis of the Stroke Health and Risk Education (SHARE) project, a behavioral stroke intervention study among a bi-ethnic population of Mexican Americans and non-Hispanic Whites, neighborhood features were collected through geocoding of addresses or zip codes to US Census Tract data.[51] Self-reported sleep duration ≤6 hours was reported in 44% of the population and was not different between the 2 ethnic groups. In a fully adjusted model, neighborhood features, including neighborhood disadvantage and violent crime, were not associated with subjective sleep duration

 Stroke. 2023;54:595–604. DOI: 10.1161/STROKEAHA.122.039524

or daytime sleepiness. However, self-reported safety was associated with a trend towards a lower odds of daytime sleepiness (odds ratio=0.85, 0.70–1.02; $P<0.10$). This finding where individual perception of neighborhood safety can have more of an impact on sleep outcomes than objective neighborhood measures has also been noted by others.[52]

The findings of SHARE were followed by a cross-sectional analysis of the JHS (Jackson Heart Study), which further evaluated in over 5000 African American participants the influence of both neighborhood characteristics and subjective perceptions of neighborhoods on self-reported sleep duration and quality.[13] Over a half of the participants reported sleeping ≤6 hours and nearly a quarter of participants reported fair to poor sleep quality. Census-derived neighborhood disadvantage was not associated with any sleep outcomes, but participants with lower SES had higher odds of self-reported long sleep. Further, perceptions of neighborhood violence were associated with subjective shorter sleep duration and poor sleep quality, even after adjustment for demographics and risk factors for poor sleep. Participants reporting high neighborhood violence had 18% higher odds of subjective short sleep and reported sleeping ≈ 10 minutes less per night on average (−9.82 minutes, −16.98 to −2.66) compared with participants who reported lower neighborhood violence. The investigators postulated that the association of lower SES with longer reported sleep and lower sleep quality may have been related to working long hours, shift work, or depressive symptoms—controlling for depressive symptoms in a secondary analysis attenuated the association between SES and sleep quality. The JHS investigators also reported similar findings as the SHARE project, in that, subjective perceptions of neighborhood disorder, including violence, were associated with sleep outcomes rather than objective measures of neighborhood disadvantage.

One of the main limitations of SHARE and JHS is the reliance of self-reported sleep, which has been shown to correlate poorly with objectively measured sleep.[53] In a cross-sectional analysis of the MESA (Multiethnic Study of Atherosclerosis) study, sleep duration and efficiency were objectively measured with 1-week wrist actigraphy among 1889 participants.[12] To assess the built environment, investigators evaluated population density, street connectivity, social engagement locations, self-reported neighborhood noise level and the Street Smart Walk Score, a composite measure assessing walkability to multiple amenities. A higher walk score was associated with increased walkability but also was theorized to be associated with decrease sleep efficiency and increase awakenings due to a greater exposure to traffic, noise, and inopportune light exposure. More social engagement destinations, increased street connectivity, higher population density, and higher Walk Scores were all associated with shorter average sleep time, even after adjustment for multiple potential influences on sleep. For example, each increase in the standard deviation of the Walk Score was associated with 23% higher odds of sleep ≤6 hours (odds ratio=1.2 [1.0–1.4]) and a mean of 8.1 minutes of less sleep (95% CI, −12.1 to −4.2). Self-reported neighborhood level noise was also associated with shorter average sleep time, increased odds of short sleep and decreased average sleep efficiency. Models that included noise level attenuated the association between measures of the built environment and sleep duration. The potential pathways for the built environment to influence sleep and have health consequences were postulated to include neighborhood noise, light exposure, traffic, air pollution, and stress. Neighborhood night-time noise, including noise from rail, aircraft, industry or road traffic, is a growing problem in modern cities that can affect sleep architecture or subjective sleep quality.[54,55] The possibility that stress could mediate the effects of the built environment on trouble falling asleep or maintaining sleep is also supported by the evidence from SHARE and JHS where perceived neighborhood safety or violence were associated with sleep duration.[13,51]

Stroke and Neighborhood Socioeconomic Status

In the nascent field of sleep health, researchers have proposed that residents living in poorer neighborhoods, including ethnic and racial minorities, are more likely to experience exposures and stressors that contribute to sleep deficiency, such as night-time noise or neighborhood disorder, which may ultimately contribute to increased cardiovascular morbidity.[6] Such a correlation between neighborhood influences and sleep may also contribute to stroke risk. In a cross-sectional analysis of the REGARDS (Reasons for Geographic and Racial Differences in Stroke) study, data on neighborhood SES was collected through geocoding of US Census Tract data among > 20 000 Black and White participants recruited from across the United States, but especially in the 8 southern states that comprise the "Stroke Belt."[56] A linear trend was noted between increasing incident ischemic stroke risk and decreasing neighborhood SES ($P<0.0001$), which remained after adjustment for demographics but was attenuated after adjustment for individual SES and traditional stroke risk factors, with greater attenuation in Black participants. The authors concluded that neighborhood SES contributes to stroke risk beyond individual SES and that traditional stroke risk factors in lower SES neighborhoods are likely in the pathway to higher stroke risk. Also, the larger mediation of the neighborhood SES effect in Black participants after adjustment for traditional stroke risk factors suggest more of a correlation between neighborhood SES and worse stroke risk profile.

One plausible mechanism through which the effects of the physical environment on sleep health could worsen traditional stroke risk factors among racial and ethnic minority groups is through effects on nocturnal blood pressure. Blood pressure dipping is the normal pattern of ≈ 10% lowering of blood pressure during sleep compared with wakefulness. Deeper and less fragmented sleep has been independently associated with dipping of diastolic and mean arterial blood pressure.[57] Conversely, increased sleep arousals resulting in poor sleep architecture, which has mostly been studied in sleep apnea, has been associated with a non-dipping blood pressure pattern.[58] Black Americans have a 67% increased risk of insomnia disorder with short sleep compared with White Americans[59] and have significantly less nocturnal dipping in systolic and diastolic blood pressure.[60] Further, blood pressure control, considered one of the most important risk factors for stroke prevention, has a population-attributable risk as high as 39% in non-Hispanic Black individuals and as high as 50% in other racial and ethnic groups.[61,62] Thus, whether neighborhood SES could worsen the stroke risk profile among low income and ethnic and racial minority groups through an increase in arousals during sleep and resulting poor sleep health requires further study.

Racial Disparities in Sleep and Psychosocial Stress

Neighborhood SES and perceived neighborhood disorder are not the only social determinants of health that may lead to disparities in sleep health and overall health. Rather, the question of how an unhealthy environment can "get under the skin"[63] or affect individual risk is likely the result of a complex interplay between multilevel social determinants. Studies of social influences on sleep health outside of the physical environment have also been explored, including psychosocial stress and perceived racial discrimination.

Given the high rates of poor sleep[35,59] and higher prevalence of stressors among Black adults,[64–66] investigators of the JHS evaluated how different measures of psychosocial stress might impair self-reported sleep duration and quality among nearly 5000 Black participants.[67] Investigators used multivariable models, adjusting for demographics, income and factors associated with sleep health, including depression. They showed that persons who reported increased psychosocial stressors, as measured by chronic stress, major life events, and acute stress, had higher odds of reporting very short sleep (<5 hours), short sleep (5–6 hours), shorter average sleep duration, and worse sleep quality (*P* for trends <0.01). When subjective sleep duration was measured as a continuous measure, the mean minutes of sleep was lower with each higher quartile of the 3 surveyed measures of psychosocial stress (*P* for trend <0.01). The investigators postulate that the mechanism through which psychosocial stressors could lead to poor sleep health is via activation of the sympathetic nervous system and hypothalamic-pituitary-adrenal axis. Further, the authors note that such stressors may be a point of intervention among Black participants for improving sleep health and downstream health outcomes.

More specific measures of psychosocial stress have also been examined as contributors to sleep health. In the cross-sectional PHRESH Zzz study (Pittsburgh Hill/Homewood Research on Neighborhoods, Sleep, and Health Study) of mostly Black adults living in urban, low-income neighborhoods, the effects of residential block quality and housing conditions were evaluated as potential influences on self-reported sleep quality and actigraphy-assessed sleep duration, sleep efficiency, and wakefulness after sleep onset.[68] After adjustment in linear regression models for sociodemographic, clinical and multiple sleep-related factors, self-reported "fair" or "poor" housing conditions was associated with 15 minutes shorter sleep duration but not associated with the likelihood of being a short sleeper (<7 hours). Distressed housing conditions were associated with similar reductions in objective sleep and significantly lower sleep quality. Objectively measured residential block quality was associated with 14 minutes shorter sleep, worse sleep efficiency, and higher wakefulness after sleep onset. Physical comfort, safety, and psychological well-being, factors critical for healthy sleep, were cited as potential mechanisms by which perceived suboptimal housing or block conditions could influence sleep. Supporting this hypothesis, shorter subjective sleep duration has also been associated with housing rent or mortgage insecurity[69] and food insecurity.[70] These findings highlight how such sleep disparities might contribute to other health disparities, including obesity-related morbidity and vascular disease, and point to interventions to lessen these disparities, such as municipal housing policies and loan programs to modernize homes.[68]

Sleep and Racial Discrimination

The effects of racial discrimination on sleep duration and quality may be one of the most disturbing associations affecting racial health disparities.[71–73] Racial and ethnic minorities have been shown in a large prospective study to be twice as likely to develop incident chronic insomnia than White individuals.[74] A cross-sectional survey study of ≈1500 patients diagnosed with insomnia disorder examined whether racial discrimination could mediate the relationship between race and insomnia severity.[19] The investigators hypothesized that more subtle and everyday forms of racism, such as microaggressions, could function as chronic stressors to trigger or exacerbate insomnia. A single-item and validated survey of self-reported racial discrimination asked participants to respond with a

 Stroke. 2023;54:595–604. DOI: 10.1161/STROKEAHA.122.039524

Likert scale to the question "Thinking about your race or ethnicity, how often have you felt treated badly or unfairly because of your race or ethnicity?" In fully adjusted multivariable models controlling for demographic and socioeconomic factors, racial discrimination mediated 57.3% of the relationship between racial minority status and insomnia severity scores. In fact, after accounting for racial discrimination, racial minority status was no longer significantly associated with insomnia severity. The indirect effect of racial discrimination on insomnia severity was also stronger in some of the non-Black racial minority groups than Black groups, accounting for 84% of the relationship in Asian and multiracial Americans. In this study, race was found to be more of a risk marker than a risk factor for insomnia severity,[19] underscoring the importance of understanding the mechanism through which racial disparities in health may arise and the potential implications about interventions to lessen such disparities.

CONCLUSIONS AND DISCUSSION

Stroke is the leading cause of serious long-term disability in the United States, and nearly a half of stroke survivors will have a fatal or non-fatal recurrent stroke over the next ten years.[75,76] Thus, along with mitigating the effects of traditional stroke risk factors, essential is a better understanding of novel lifestyle factors that may influence stroke risk, including sleep health, and the multilevel social determinants of health. Although sleep health may not be considered a typical stroke risk factor, observational studies to date support a multifaceted role of sleep health in stroke risk and overall well-being.

Given the observational study design of most of the studies examining poor sleep health and increased risk of stroke, a causal relationship can be hypothesized but cannot be established. Intervention studies could provide evidence linking poor sleep health as a causal risk factor for vascular disease, though such research has lagged behind observation studies in evaluating this association.[2] However, intervention studies focused on improving the vascular consequences of poor sleep health have demonstrated the benefits of habitual sleep extension on blood pressure lowering,[77] improved glucose metabolism,[78] and insulin sensitivity.[79] Another challenge in the field of sleep health will be addressing the implementation gap between the scientific knowledge regarding sleep health and the development and implementation of effective interventions at the individual level but also at the community and societal levels.[2,80]

At the individual level, screening for poor sleep habits by clinical providers may provide opportunities for healthy sleep interventions, including recommendations for personalized "sleep hygiene" behaviors, circadian health interventions, in-person and Web-based coaching, and use of mobile technology.[2,81] Another individual sleep health intervention to be further examined or implemented is health care screening for social determinants of health, including housing or food insecurity and psychosocial stressors such as perceived racial discrimination. Screening for social determinants of health are not typically performed by clinical providers,[82] though awareness of such contributors to sleep health could lead to culturally sensitive interventions in those at risk for stroke and potentially modify downstream health outcomes. At the community level,[2] interventional research or public policy to improve sleep health could focus on changes in the workplace environment,[83] school start times,[84] or health care systems.[85] Areas of continued study should also include understanding the role of both historical and contemporaneous neighborhood characteristics on the relationship between sleep health and stroke risk. Community-based studies of regional differences in stroke risk, for example, have found that childhood residency and proportion of life in the Stroke Belt region of the southeastern US conferred a high stroke risk in middle or old age even among those who no longer lived in this area.[86,87] At the societal level, more research is needed to understand how changes in public policy can influence social factors that affect sleep health, stroke risk and the relationship between the 2. For example, quasi-experimental designs have been utilized to evaluate the impact of policy change designed to improve neighborhood disadvantage on overall poor health outcomes.[88] More work is also needed to assess how policies to address problems in the built environment might improve sleep health; for example, ordinances to reduce noise[89] or initiatives to reduce light pollution.[55]

Ultimately, the goal of increased scholarship in the relationship between sleep health and stroke risk would be to lead to the development of interventions to address these disparities. Sleep and public health researchers can play an important role in the promotion of evidence-based interventions for sleep health targeted at patients, educators, industry partners, professional societies, and funding-decision or policy makers.[80] Given the potential for vascular disease prevention and improved patient outcomes, the public health implications of such sleep health interventions could be substantial, especially for socioeconomically disadvantaged groups and ethnic and racial minorities.

ARTICLE INFORMATION

Affiliations

Department of Neurology (S.P.K., B.L.T., W.T.L.) and Department of Epidemiology (W.T.L.), University of Washington, Seattle. Department of Medicine, University of California, Los Angeles (A.F.B.).

Sources of Funding

Dr Brown received funding from NIH/NIMHD: #1 P50 MD017366-01 (Title: UCLA-UCI Center for Eliminating Cardio-Metabolic Disparities in Multi-Ethnic Populations [UC END-DISPARITIES]).

Disclosures

None.

TOPICAL REVIEW

Supplemental Material

Table S1
Figures S1–S2

REFERENCES

1. Buysse DJ. Sleep health: can we define it? Does it matter? *Sleep*. 2014;37:9–17. doi: 10.5665/sleep.3298
2. Hale L, Troxel W, Buysse DJ. Sleep health: an opportunity for public health to address health equity. *Annu Rev Public Health*. 2020;41:81–99. doi: 10.1146/annurev-publhealth-040119-094412
3. Khot SP, Morgenstern LB. Sleep and stroke. *Stroke*. 2019;50:1612–1617. doi: 10.1161/STROKEAHA.118.023553
4. Cappuccio FP, Cooper D, D'Elia L, Strazzullo P, Miller MA. Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. *Eur Heart J*. 2011;32:1484–1492. doi: 10.1093/eurheartj/ehr007
5. Yaffe K, Nasrallah I, Hoang TD, Lauderdale DS, Knutson KL, Carnethon MR, Launer LJ, Lewis CE, Sidney S. Sleep duration and white matter quality in middle-aged adults. *Sleep*. 2016;39:1743–1747. doi: 10.5665/sleep.6104
6. Jackson CL, Redline S, Emmons KM. Sleep as a potential fundamental contributor to disparities in cardiovascular health. *Annu Rev Public Health*. 2015;36:417–440. doi: 10.1146/annurev-publhealth-031914-122838
7. Johnson KG, Johnson DC. Frequency of sleep apnea in stroke and TIA patients: a meta-analysis. *J Clin Sleep Med*. 2010;6:131–137. doi: 10.5664/jcsm.27760
8. Redline S, Yenokyan G, Gottlieb DJ, Shahar E, O'Connor GT, Resnick HE, Diener-West M, Sanders MH, Wolf PA, Geraghty EM, et al. Obstructive sleep apnea-hypopnea and incident stroke: the Sleep Heart Health Study. *Am J Respir Crit Care Med*. 2010;182:269–277. doi: 10.1164/rccm.200911-1746OC
9. Dong JY, Zhang YH, Qin LQ. Obstructive sleep apnea and cardiovascular risk: meta-analysis of prospective cohort studies. *Atherosclerosis*. 2013;229:489–495. doi: 10.1016/j.atherosclerosis.2013.04.026
10. Martinez-Garcia MA, Campos-Rodriguez F, Soler-Cataluna JJ, Catalan-Serra P, Roman-Sanchez P, Montserrat JM. Increased incidence of nonfatal cardiovascular events in stroke patients with sleep apnoea: effect of CPAP treatment. *Eur Respir J*. 2012;39:906–912. doi: 10.1183/09031936.00011311
11. Brown DL, Shafie-Khorassani F, Kim S, Chervin RD, Case E, Morgenstern LB, Yadollahi A, Tower S, Lisabeth LD. Sleep-disordered breathing is associated with recurrent ischemic stroke. *Stroke*. 2019;50:571–576. doi: 10.1161/STROKEAHA.118.023807
12. Johnson DA, Hirsch JA, Moore KA, Redline S, Diez Roux AV. Associations between the built environment and objective measures of sleep: the Multi-Ethnic Study of Atherosclerosis. *Am J Epidemiol*. 2018;187:941–950. doi: 10.1093/aje/kwx302
13. Johnson DA, Lisabeth L, Hickson D, Johnson-Lawrence V, Samdarshi T, Taylor H, Diez Roux AV. The social patterning of sleep in African Americans: associations of socioeconomic position and neighborhood characteristics with sleep in the Jackson Heart Study. *Sleep*. 2016;39:1749–1759. doi: 10.5665/sleep.6106
14. Chen JC, Brunner RL, Ren H, Wassertheil-Smoller S, Larson JC, Levine DW, Allison M, Naughton MJ, Stefanick ML. Sleep duration and risk of ischemic stroke in postmenopausal women. *Stroke*. 2008;39:3185–3192. doi: 10.1161/STROKEAHA.108.521773
15. Qureshi AI, Giles WH, Croft JB, Bliwise DL. Habitual sleep patterns and risk for stroke and coronary heart disease: a 10-year follow-up from NHANES I. *Neurology*. 1997;48:904–911. doi: 10.1212/wnl.48.4.904
16. Vernon MK, Dugar A, Revicki D, Treglia M, Buysse D. Measurement of non-restorative sleep in insomnia: a review of the literature. *Sleep Med Rev*. 2010;14:205–212. doi: 10.1016/j.smrv.2009.10.002
17. Matsumoto T, Chin K. Prevalence of sleep disturbances: sleep disordered breathing, short sleep duration, and non-restorative sleep. *Respir Investig*. 2019;57:227–237. doi: 10.1016/j.resinv.2019.01.008
18. Bertisch SM, Pollock BD, Mittleman MA, Buysse DJ, Bazzano LA, Gottlieb DJ, Redline S. Insomnia with objective short sleep duration and risk of incident cardiovascular disease and all-cause mortality: Sleep Heart Health Study. *Sleep*. 2018;41:zsy047. doi: 10.1093/sleep/zsy047
19. Cheng P, Cuellar R, Johnson DA, Kalmbach DA, Joseph CL, Cuamatzi Castelan A, Sagong C, Casement MD, Drake CL. Racial discrimination as a mediator of racial disparities in insomnia disorder. *Sleep Health*. 2020;6:543–549. doi: 10.1016/j.sleh.2020.07.007
20. Mullington JM, Haack M, Toth M, Serrador JM, Meier-Ewert HK. Cardiovascular, inflammatory, and metabolic consequences of sleep deprivation. *Prog Cardiovasc Dis*. 2009;51:294–302. doi: 10.1016/j.pcad.2008.10.003
21. Patel SR, Hu FB. Short sleep duration and weight gain: a systematic review. *Obesity (Silver Spring)*. 2008;16:643–653. doi: 10.1038/oby.2007.118
22. Gottlieb DJ, Redline S, Nieto FJ, Baldwin CM, Newman AB, Resnick HE, Punjabi NM. Association of usual sleep duration with hypertension: the sleep heart health study. *Sleep*. 2006;29:1009–1014. doi: 10.1093/sleep/29.8.1009
23. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis. *Diabetes Care*. 2010;33:414–420. doi: 10.2337/dc09-1124
24. Seravalle G, Mancia G, Grassi G. Sympathetic nervous system, sleep, and hypertension. *Curr Hypertens Rep*. 2018;20:74. doi: 10.1007/s11906-018-0874-y
25. Basta M, Chrousos GP, Vela-Bueno A, Vgontzas AN. Chronic insomnia and stress system. *Sleep Med Clin*. 2007;2:279–291. doi: 10.1016/j.jsmc.2007.04.002
26. Gangwisch JE. A review of evidence for the link between sleep duration and hypertension. *Am J Hypertens*. 2014;27:1235–1242. doi: 10.1093/ajh/hpu071
27. O'Halloran D, O'Boyle C, Doherty L. Poor sleep associated with clinically severe obesity is independent of OSA status. *Obes Surg*. 2021;31:4734–4740. doi: 10.1007/s11695-021-05588-3
28. Watson NF, Badr MS, Belenky G, Bliwise DL, Buxton OM, Buysse D, Dinges DF, Gangwisch J, Grandner MA, Kushida C; Consensus Conference Panel. 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;38:1161–1183. doi: 10.5665/sleep.4886
29. Hirshkowitz M, Whiton K, Albert SM, Alessi C, Bruni O, DonCarlos L, Hazen N, Herman J, Katz ES, Kheirandish-Gozal L, et al. National Sleep Foundation's sleep time duration recommendations: methodology and results summary. *Sleep Health*. 2015;1:40–43. doi: 10.1016/j.sleh.2014.12.010
30. Krueger PM, Friedman EM. Sleep duration in the United States: a cross-sectional population-based study. *Am J Epidemiol*. 2009;169:1052–1063. doi: 10.1093/aje/kwp023
31. Ford ES, Cunningham TJ, Croft JB. Trends in self-reported sleep duration among US adults from 1985 to 2012. *Sleep*. 2015;38:829–832. doi: 10.5665/sleep.4684
32. Altman NG, Izci-Balserak B, Schopfer E, Jackson N, Rattanaumpawan P, Gehrman PR, Patel NP, Grandner MA. Sleep duration versus sleep insufficiency as predictors of cardiometabolic health outcomes. *Sleep Med*. 2012;13:1261–1270. doi: 10.1016/j.sleep.2012.08.005
33. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. *Sleep*. 2010;33:585–592. doi: 10.1093/sleep/33.5.585
34. Whinnery J, Jackson N, Rattanaumpawan P, Grandner MA. Short and long sleep duration associated with race/ethnicity, sociodemographics, and socioeconomic position. *Sleep*. 2014;37:601–611. doi: 10.5665/sleep.3508
35. Pigeon WR, Heffner K, Duberstein P, Fiscella K, Moynihan J, Chapman BP. Elevated sleep disturbance among blacks in an urban family medicine practice. *J Am Board Fam Med*. 2011;24:161–168. doi: 10.3122/jabfm.2011.02.100028
36. Chapman DP, Croft JB, Liu Y, Perry GS, Presley-Cantrell LR, Ford ES. Excess frequent insufficient sleep in American Indians/Alaska Natives. *J Environ Public Health*. 2013;2013:259645. doi: 10.1155/2013/259645
37. Cox AM, McKevitt C, Rudd AG, Wolfe CD. Socioeconomic status and stroke. *Lancet Neurol*. 2006;5:181–188. doi: 10.1016/S1474-4422(06)70351-9
38. Stansbury JP, Jia H, Williams LS, Vogel WB, Duncan PW. Ethnic disparities in stroke: epidemiology, acute care, and postacute outcomes. *Stroke*. 2005;36:374–386. doi: 10.1161/01.STR.0000153065.39325.fd
39. Sheriff F, Xu H, Maud A, Gupta V, Vellipuram A, Fonarow GC, Matsouaka RA, Xian Y, Reeves M, Smith EE, et al. Temporal trends in racial and ethnic disparities in endovascular therapy in acute ischemic stroke. *J Am Heart Assoc*. 2022;11:e023212. doi: 10.1161/JAHA.121.023212
40. Howard G, Safford MM, Moy CS, Howard VJ, Kleindorfer DO, Unverzagt FW, Soliman EZ, Flaherty ML, McClure LA, Lackland DT, et al. Racial differences in the incidence of cardiovascular risk factors in older black and white adults. *J Am Geriatr Soc*. 2017;65:83–90. doi: 10.1111/jgs.14472
41. Liao Y, Greenlund KJ, Croft JB, Keenan NL, Giles WH. Factors explaining excess stroke prevalence in the US stroke belt. *Stroke*. 2009;40:3336–3341. doi: 10.1161/STROKEAHA.109.561688

42. Kapral MK, Fang J, Chan C, Alter DA, Bronskill SE, Hill MD, Manuel DG, Tu JV, Anderson GM. Neighborhood income and stroke care and outcomes. *Neurology.* 2012;79:1200–1207. doi: 10.1212/WNL.0b013e31826aac9b
43. Brown AF, Liang LJ, Vassar SD, Merkin SS, Longstreth WT Jr, Ovbiagele B, Yan T, Escarce JJ. Neighborhood socioeconomic disadvantage and mortality after stroke. *Neurology.* 2013;80:520–527. doi: 10.1212/WNL.0b013e31828154ae
44. Wang S, Zhai H, Wei L, Shen B, Wang J. Socioeconomic status predicts the risk of stroke death: a systematic review and meta-analysis. *Prev Med Rep.* 2020;19:101124. doi: 10.1016/j.pmedr.2020.101124
45. Howard VJ, Kleindorfer DO, Judd SE, McClure LA, Safford MM, Rhodes JD, Cushman M, Moy CS, Soliman EZ, Kissela BM, et al. Disparities in stroke incidence contributing to disparities in stroke mortality. *Ann Neurol.* 2011;69:619–627. doi: 10.1002/ana.22385
46. O'Donnell D, Silva EJ, Munch M, Ronda JM, Wang W, Duffy JF. Comparison of subjective and objective assessments of sleep in healthy older subjects without sleep complaints. *J Sleep Res.* 2009;18:254–263. doi: 10.1111/j.1365-2869.2008.00719.x
47. Unruh ML, Redline S, An MW, Buysse DJ, Nieto FJ, Yeh JL, Newman AB. Subjective and objective sleep quality and aging in the Sleep Heart Health Study. *J Am Geriatr Soc.* 2008;56:1218–1227. doi: 10.1111/j.1532-5415.2008.01755.x
48. Warnecke RB, Oh A, Breen N, Gehlert S, Paskett E, Tucker KL, Lurie N, Rebbeck T, Goodwin J, Flack J. Approaching health disparities from a population perspective: the National Institutes of Health Centers for Population Health and Health Disparities. *Am J Public Health.* 2008;98:1608–1615. doi: 10.2105/AJPH.2006.102525
49. Billings ME, Hale L, Johnson DA. Physical and social environment relationship with sleep health and disorders. *Chest.* 2020;157:1304–1312. doi: 10.1016/j.chest.2019.12.002
50. Billings ME, Gold D, Szpiro A, Aaron CP, Jorgensen N, Gassett A, Leary PJ, Kaufman JD, Redline SR. The association of ambient air pollution with sleep apnea: the Multi-Ethnic Study of Atherosclerosis. *Ann Am Thorac Soc.* 2019;16:363–370. doi: 10.1513/AnnalsATS.201804-248OC
51. Johnson DA, Brown DL, Morgenstern LB, Meurer WJ, Lisabeth LD. The association of neighborhood characteristics with sleep duration and daytime sleepiness. *Sleep Health.* 2015;1:148–155. doi: 10.1016/j.sleh.2015.06.002
52. Hill TD, Trinh HN, Wen M, Hale L. Perceived neighborhood safety and sleep quality: a global analysis of six countries. *Sleep Med.* 2016;18:56–60. doi: 10.1016/j.sleep.2014.12.003
53. Lauderdale DS, Knutson KL, Yan LL, Liu K, Rathouz PJ. Self-reported and measured sleep duration: how similar are they?. *Epidemiology.* 2008;19:838–845. doi: 10.1097/EDE.0b013e318187a7b0
54. Halperin D. Environmental noise and sleep disturbances: a threat to health?. *Sleep Sci.* 2014;7:209–212. doi: 10.1016/j.slsci.2014.11.003
55. Hu K, Li W, Zhang Y, Chen H, Bai C, Yang Z, Lorenz T, Liu K, Shirai K, Song J, et al. Association between outdoor artificial light at night and sleep duration among older adults in China: a cross-sectional study. *Environ Res.* 2022;212:113343. doi: 10.1016/j.envres.2022.113343
56. Howard VJ, McClure LA, Kleindorfer DO, Cunningham SA, Thrift AG, Diez Roux AV, Howard G. Neighborhood socioeconomic index and stroke incidence in a national cohort of blacks and whites. *Neurology.* 2016;87:2340–2347. doi: 10.1212/WNL.0000000000003299
57. Loredo JS, Nelesen R, Ancoli-Israel S, Dimsdale JE. Sleep quality and blood pressure dipping in normal adults. *Sleep.* 2004;27:1097–1103. doi: 10.1093/sleep/27.6.1097
58. Bradley TD, Floras JS. Obstructive sleep apnoea and its cardiovascular consequences. *Lancet.* 2009;373:82–93. doi: 10.1016/S0140-6736(08)61622-0
59. Kalmbach DA, Pillai V, Arnedt JT, Drake CL. DSM-5 insomnia and short sleep: comorbidity landscape and racial disparities. *Sleep.* 2016;39:2101–2111. doi: 10.5665/sleep.6306
60. Profant J, Dimsdale JE. Race and diurnal blood pressure patterns. A review and meta-analysis. *Hypertension.* 1999;33:1099–1104. doi: 10.1161/01.hyp.33.5.1099
61. Clark D 3rd, Colantonio LD, Min YI, Hall ME, Zhao H, Mentz RJ, Shimbo D, Ogedegbe G, Howard G, Levitan EB, et al. Population-attributable risk for cardiovascular disease associated with hypertension in black adults. *JAMA Cardiol.* 2019;4:1194–1202. doi: 10.1001/jamacardio.2019.3773
62. Willey JZ, Moon YP, Kahn E, Rodriguez CJ, Rundek T, Cheung K, Sacco RL, Elkind MS. Population attributable risks of hypertension and diabetes for cardiovascular disease and stroke in the Northern Manhattan Study. *J Am Heart Assoc.* 2014;3:e001106. doi: 10.1161/JAHA.114.001106
63. Taylor SE, Repetti RL, Seeman T. Health psychology: what is an unhealthy environment and how does it get under the skin?. *Annu Rev Psychol.* 1997;48:411–447. doi: 10.1146/annurev.psych.48.1.411
64. Hatch SL, Dohrenwend BP. Distribution of traumatic and other stressful life events by race/ethnicity, gender, SES and age: a review of the research. *Am J Community Psychol.* 2007;40:313–332. doi: 10.1007/s10464-007-9134-z
65. Williams DR, Mohammed SA. Discrimination and racial disparities in health: evidence and needed research. *J Behav Med.* 2009;32:20–47. doi: 10.1007/s10865-008-9185-0
66. Williams DR, Mohammed SA, Leavell J, Collins C. Race, socioeconomic status, and health: complexities, ongoing challenges, and research opportunities. *Ann N Y Acad Sci.* 2010;1186:69–101. doi: 10.1111/j.1749-6632.2009.05339.x
67. Johnson DA, Lisabeth L, Lewis TT, Sims M, Hickson DA, Samdarshi T, Taylor H, Roux AV. The contribution of psychosocial stressors to sleep among African Americans in the Jackson Heart Study. *Sleep.* 2016;39:1411–1419. doi: 10.5665/sleep.5974
68. Troxel WM, Haas A, Ghosh-Dastidar B, Holliday SB, Richardson AS, Schwartz H, Gary-Webb TL, Hale L, Buysse DJ, Buman MP, et al. Broken windows, broken ZZs: poor housing and neighborhood conditions are associated with objective measures of sleep health. *J Urban Health.* 2020;97:230–238. doi: 10.1007/s11524-019-00418-5
69. Bozick R, Troxel WM, Karoly LA. Housing insecurity and sleep among welfare recipients in California. *Sleep.* 2021;44:zsab005. doi: 10.1093/sleep/zsab005
70. Liu Y, Njai RS, Greenlund KJ, Chapman DP, Croft JB. Relationships between housing and food insecurity, frequent mental distress, and insufficient sleep among adults in 12 US states, 2009. *Prev Chronic Dis.* 2014;11:E37. doi: 10.5888/pcd11.130334
71. Bethea TN, Zhou ES, Schernhammer ES, Castro-Webb N, Cozier YC, Rosenberg L. Perceived racial discrimination and risk of insomnia among middle-aged and elderly black women. *Sleep.* 2020;43:zsz208. doi: 10.1093/sleep/zsz208
72. Grandner MA, Hale L, Jackson N, Patel NP, Gooneratne NS, Troxel WM. Perceived racial discrimination as an independent predictor of sleep disturbance and daytime fatigue. *Behav Sleep Med.* 2012;10:235–249. doi: 10.1080/15402002.2012.654548
73. Lee S, Chang AM, Buxton OM, Jackson CL. Various types of perceived job discrimination and sleep health among working women: findings from the Sister Study. *Am J Epidemiol.* 2020;189:1143–1153. doi: 10.1093/aje/kwaa075
74. Singareddy R, Vgontzas AN, Fernandez-Mendoza J, Liao D, Calhoun S, Shaffer ML, Bixler EO. Risk factors for incident chronic insomnia: a general population prospective study. *Sleep Med.* 2012;13:346–353. doi: 10.1016/j.sleep.2011.10.033
75. Hardie K, Hankey GJ, Jamrozik K, Broadhurst RJ, Anderson C. Ten-year risk of first recurrent stroke and disability after first-ever stroke in the Perth Community Stroke Study. *Stroke.* 2004;35:731–735. doi: 10.1161/01.STR.0000116183.50167.D9
76. Virani SS, Alonso A, Aparicio HJ, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, Chamberlain AM, Cheng S, Delling FN, et al. Heart disease and stroke statistics-2021 update: a report from the American Heart Association. *Circulation.* 2021;143:e254–e743. doi: 10.1161/CIR.0000000000000950
77. Haack M, Serrador J, Cohen D, Simpson N, Meier-Ewert H, Mullington JM. Increasing sleep duration to lower beat-to-beat blood pressure: a pilot study. *J Sleep Res.* 2013;22:295–304. doi: 10.1111/jsr.12011
78. So-Ngern A, Chirakalwasan N, Saetung S, Chanprasertyothin S, Thakkinstian A, Reutrakul S. Effects of two-week sleep extension on glucose metabolism in chronically sleep-deprived individuals. *J Clin Sleep Med.* 2019;15:711–718. doi: 10.5664/jcsm.7758
79. Leproult R, Deliens G, Gilson M, Peigneux P. Beneficial impact of sleep extension on fasting insulin sensitivity in adults with habitual sleep restriction. *Sleep.* 2015;38:707–715. doi: 10.5665/sleep.4660
80. Parthasarathy S, Carskadon MA, Jean-Louis G, Owens J, Bramoweth A, Combs D, Hale L, Harrison E, Hart CN, Hasler BP, et al. Implementation of sleep and circadian science: recommendations from the Sleep Research Society and National Institutes of Health workshop. *Sleep.* 2016;39:2061–2075. doi: 10.5665/sleep.6300
81. Irish LA, Kline CE, Gunn HE, Buysse DJ, Hall MH. The role of sleep hygiene in promoting public health: a review of empirical evidence. *Sleep Med Rev.* 2015;22:23–36. doi: 10.1016/j.smrv.2014.10.001
82. Andermann A. Screening for social determinants of health in clinical care: moving from the margins to the mainstream. *Public Health Rev.* 2018;39:19. doi: 10.1186/s40985-018-0094-7

83. Olson R, Crain TL, Bodner TE, King R, Hammer LB, Klein LC, Erickson L, Moen P, Berkman LF, Buxton OM. A workplace intervention improves sleep: results from the randomized controlled work, family, and health study. *Sleep Health*. 2015;1:55–65. doi: 10.1016/j.sleh.2014.11.003
84. Barnes M, Davis K, Mancini M, Ruffin J, Simpson T, Casazza K. Setting adolescents up for success: promoting a policy to delay high school start times. *J Sch Health*. 2016;86:552–557. doi: 10.1111/josh.12405
85. Redeker NS. Sleep in acute care settings: an integrative review. *J Nurs Scholarsh*. 2000;32:31–38. doi: 10.1111/j.1547-5069.2000.00031.x
86. Glymour MM, Avendano M, Berkman LF. Is the "stroke belt" worn from childhood?: Risk of first stroke and state of residence in childhood and adulthood. *Stroke*. 2007;38:2415–2421. doi: 10.1161/STROKEAHA.107.482059
87. Howard VJ, McClure LA, Glymour MM, Cunningham SA, Kleindorfer DO, Crowe M, Wadley VG, Peace F, Howard G, Lackland DT. Effect of duration and age at exposure to the stroke belt on incident stroke in adulthood. *Neurology*. 2013;80:1655–1661. doi: 10.1212/WNL.0b013e3182904d59
88. Mehdipanah R, Rodriguez-Sanz M, Malmusi D, Muntaner C, Diez E, Bartoll X, Borrell C. The effects of an urban renewal project on health and health inequalities: a quasi-experimental study in Barcelona. *J Epidemiol Community Health*. 2014;68:811–817. doi: 10.1136/jech-2013-203434
89. Wojciechowska W, Januszewicz A, Drozdz T, Rojek M, Baczalska J, Terlecki M, Kurasz K, Olszanecka A, Smólski M, Prejbisz A, et al. Blood pressure and arterial stiffness in association with aircraft noise exposure: long-term observation and potential effect of COVID-19 lockdown. *Hypertension*. 2022;79:325–334. doi: 10.1161/HYPERTENSIONAHA.121.17704