Stroke

TOPICAL REVIEW

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

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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

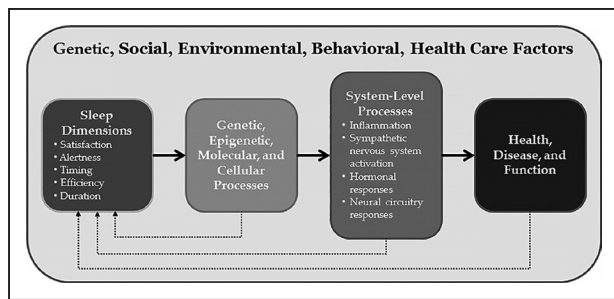*Stroke* is available at www.ahajournals.org/journal/str

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,

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

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

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

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Disclosures

None.

Supplemental Material

Table S1

Figures S1–S2

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