Chapter 3
Sleep Health among Racial/Ethnic groups and Strategies to achieve Sleep Health Equity

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Keywords Sleep quality · Rapid eye movement · Insomnia · Circadian rhythms · Social jetlag · Non-rapid eye movement (NREM) · Sleep architecture · Thyromental angle

Introduction

Relative to Whites, racial/ethnic minorities are more likely to experience a higher burden of poor health, chronic disease, accelerated aging, and premature/excess deaths [1–6]. These health burdens can be attributed to several biological, psychosocial, and environmental factors and mechanisms. Notable biological explanations include, but are not limited to, advanced cell aging, DNA methylation, telomerization of cells, and multimorbidity [2, 7–14]. However, the pathogenesis of poor health, accelerated aging, and disease burden among racial/ethnic minorities is not solely a biological process; it also occurs epigenetically where chronic exposure to

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M. S. Badr, J. L. Martin (eds.), *Essentials of Sleep Medicine*,
Respiratory Medicine, https://doi.org/10.1007/978-3-030-93739-3_3

noxious exogenous factors cause disease by introducing heritable changes in genetic expression or chromosomal function into the biology of individuals to cause disease [11, 12, 15–17]. Growing evidence highlights that the burden of poor health, chronic disease, and accelerated cellular aging among racial/ ethnic minorities is observed across the lifespan, suggesting that the roots of poor health begin as early as the in-utero period and childhood [18]. However, extant etiological frameworks to explain the burden of poor health, chronic disease, accelerated aging, and premature/excess deaths heavily emphasize proximal and exogenous effects of life stressors and exclude potential distal and upstream factors such as the cumulative effect of prejudice and discrimination, as well as environmental (noxious noise, light, and air quality), political (taxes, policies, governmental programs, and nativity/citizen status) [19], and social determinants of health (poor access to health services, socioeconomic status, neighborhoods, and education) [20].

Although it is difficult to identify a single cause for the burden of poor health outcomes, chronic disease, accelerated aging, and premature/excess deaths, there are some factors that may offer upstream (etiology) and downstream (consequences) insights into the chronicity, pervasiveness, and ubiquity of poor health among racial/ethnic minorities. Growing evidence points to sleep health (duration, sleep disorders, efficiency, sleep quality, sleepiness (alertness), and timing/chronotype) as being one such factor that might provide a unique upstream and downstream explanation for the life-course burden of poor health, chronic disease, accelerated aging, and premature/excess deaths among racial/ethnic minorities [21–25].

Sleep disruptions represent both a cause and consequence of poor health outcomes in racial/ethnic minorities. Mounting evidence indicates that racial/ethnic groups are unequally burdened by a wide range of sleep disruptions and poor sleep health outcomes, which lead to further disparities in cardiovascular disease, cancer, dementia, and mental health outcomes [21–28]. Although the burden of poor sleep health outcomes affects most racial/ethnic groups, relative to Whites, their manifestation and causes at the population level are specific to each group. For example, Black and Latino children and adults alike have a predominantly higher risk for poor sleep health (inconsistent sleep schedules, insufficient sleep duration, sleep-disordered breathing problems, and daytime sleepiness) compared to their White counterparts. It is likely that poor sleep health, experienced as a child, has residual and insidious effects on later life, thus increasing likelihood of debilitating poor sleep and adverse functional and health outcomes, such as poor cognitive performance, car accidents and occupational accidents, cardiovascular disease, diabetes, and mental health, all of which are higher among racial/ethnic minorities compared to Whites [29, 30].

To better understanding sleep health burden among racial/ethnic groups, we will describe the prevalence and burden of sleep health parameters: (1) sleep duration, (2) sleep disorders, (3) sleep timing/chronotype, (4) sleep architecture, (5) sleep quality, (6) sleepiness/alertness, and (7) sleep efficiency (See Fig. 3.1). Then, we will describe upstream processes and causes of poor sleep health parameters among racial/ethnic groups, which can be attributed to a variety of biological (circadian rhythm), behavioral (diet and exercise mental health), psychosocial (stress, mental

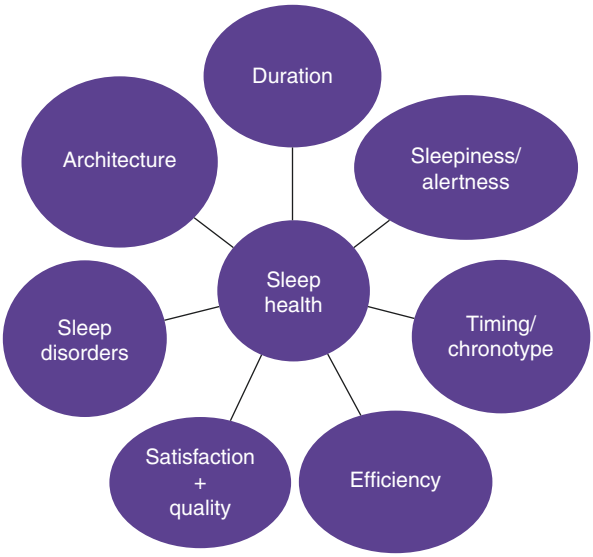

Fig. 3.1 Sleep health components diagram

health, poverty, social demands), and environmental factors (noise and light), which we describe in detail. Lastly, we will describe the downstream processes and consequences of poor sleep health among racial/ethnic groups, which include but are not limited to a variety of adverse health outcomes, chronic diseases (cardiovascular disease, diabetes, and mental health), and poor functional and performance outcomes.

Prevalence and Burden of Poor Sleep Health

Sleep health is characterized by seven sleep parameters: (1) sleep duration, (2) sleep disorders, (3) sleep timing/chronotype, (4) sleep architecture (sleep stages divided into rapid eye movement (REM) and non-REM sleep), (5) sleep quality, (6) sleepiness, and (7) sleep efficiency [31]. Mounting evidence highlight an alarming trend of sleep health disparities across certain demographic groups, most notably among racial/ethnic minorities who compared to their White counterparts experience significant burden in all seven sleep parameters which have been attributed to several adverse health and functional outcomes and provide some explanation for elevated and high burden of certain chronic diseases, such as cardiovascular disease, mental health, and dementia. From this growing and heterogenous evidence, there is an emerging coalescing definition of sleep health disparity, which the NIH describes as any difference in one or more dimensions of sleep health (regularity, quality, alertness, timing, efficiency, and duration)—on a consistent basis—that adversely affects designated disadvantaged populations [29]. Although this definition of sleep health disparity is not final or comprehensive, it represents an excellent start and working

definition that provides the critical lens through which one can identify, define, measure, and study the drivers and consequences of sleep health disparity. Therefore, we use this definition of sleep health disparity as the lens to identify sleep health disparities among racial/ethnic groups for the current book chapter. However, to better understand sleep health disparity, it is important to go beyond identifying differences (which is often restricted to numerical difference), and seek to understand the fundamental causes and consequences of these differences.

Therefore, for the current chapter, we define sleep health disparity as differences that are due to deeply entrenched cause[s] (biological, psychosocial, and environmental) and downstream consequences that even the counterfactual cannot escape. For example, sleep health disparity exists because both poor and wealthy racial/ethnic minorities are more burdened by poor sleep health outcomes, compared to their poor and wealthy White counterparts (even when comparing a wealthy minority with a poor White individual). This evidence should not be misunderstood as a positing and privileging of ontogeny/biological causes over social causes but rather an acknowledgment of the syndemic and epigenetic etiology of sleep health disparity. In fact, sleep health disparity may be due to omni-directional relationships among latent hard-wired biological factors, noxious psychosocial and environmental terroir and contexts, and defunct system-level factors that would normally ameliorate health risk (such as the inability of our poor healthcare system to address sleep health disparities and its consequences or poor labor policies that prevent individuals from earning a living wage causing poorer populations to work multiple jobs that induce stress and disrupt sleep health). For the sections below, we establish: (1) sleep health disparities by describing numerical differences across all sleep health parameters (highlighting higher burden, prevalence rates, and likelihoods among racial/ethnic minorities compared to their counterfactual counterparts), (2) biological, psychosocial, and environmental antecedents and causes of sleep health differences, and (3) functional and health consequences of sleep health differences among racial/ethnic minorities (See Table 3.1).

Sleep Duration

There are significant differences in sleep duration and total sleep time among several demographic groups (sex, geographic, and socioeconomic status). The most compelling and robust evidence for group-based differences in sleep duration are observed among racial/ethnic minorities and so for the current chapter we focus only on racial/ethnic groups. Racial/ethnic differences in sleep duration have been noticed as early as childhood and persist throughout the lifespan to adulthood. Regardless of age group, racial/ethnic minorities do not receive adequate sleep duration for their age sleep. Although the American Academy of Sleep Medicine recommends that 3–5-year-old children receive on average 10–13 hours (including naps) daily, 9–12 hours for 9–12-year-old children, and 7–9 hours for adults, racial/ethnic minorities consistently experience insufficient sleep duration (See Table 3.1).

Table 3.1 Racial-ethnic disparities in health outcomes in selected sleep dimensions compared to White adults

	Sleep duration	Sleep disorders	Chrono type-circadian rhythm	Sleep architecture	Social jet lag-excessive sleepiness	Sleep quality
African-American/ Black	↓ 12–14,29,30–33	↓ 6,19,31,32,44–46	↓ 50,52,53	↓ 64,21	↓ 23,43	↓ 44,57,58,59
Hispanics-Latinos	↓ 17,26,38	↓ 20,31,35,47,49	↓ IES	↓ 64	↓ 43	↓ 57
Asian	↓ 12	31,20	IES	IES	IES	IES
Native Hawaiian and Pacific Islander	↓ 12	44	IES	64	26	IES

Note: The direction of the arrow refers to the direction of how these groups are more at risk to experience sleep disparities in the selected categories (e.g., lower or higher)
Abbreviations: *MR* mixed results, IES insufficient evidence in sleep

Chronic insufficient sleep duration (such as short sleep duration <7 hours) has been linked with increased risk for cognitive impairment, occupational hazards, mistakes, poor cardiovascular health and disease, mental illness, dementia, and cancer [32–36].

Based on data from the National Health and Nutrition Examination Survey (NHANES), short sleep duration is highly prevalent in the United States with a conservative overall estimate of 37.1% across the lifespan. However, these same data reveal that the greatest burden of short sleep duration is experienced among middle age individuals: (1) 20–39 years of age (37.0%); (2) 40–59 years (40.3%); and 60 years and older (32.0%), highlighting severe sleep deprivation across all age groups [37]. Stratifying these results by race/ethnicity highlights the fact that among children 6 months and 2 years old, only 6% of Black children slept the recommended amount of 12 hours daily, while 83% of White children slept at least 12 hours daily. Similar trends have been observed among adolescents, where Asians (76%) and Blacks (71%) had the highest rates of short sleep duration/insufficient sleep relative to Whites (68%), except Latinx (67%). Similarly among adults, Native Hawaiian-Pacific Islanders (46.3%); Blacks (45.8%); Other Multiracial (44.3%); American Indian-Alaska Native (40.4%); Asian (37.5%); and Latins (34.5%) all have higher prevalence of short/insufficient sleep relative to Whites (33.4%).

Sleep health disparities among minorities are not limited to adult populations only. A review of 23 studies investigated racial/ethnic sleep health disparities among American minority youth between the ages of 6–19 years and found that white youth (adolescents) had more sufficient sleep compared to racial/ethnic minorities, most notably Blacks and Hispanics/Latinos. Blacks had overall shorter sleep

duration and later bedtimes than Hispanics/Latinos [38]. Black and Hispanic youth also spent more time traveling to school, had earlier start times, and spent more time watching television, more likely to share a bedroom and partake in regular naps. Evidently, napping decreased with age, and some studies have shown that Blacks and Hispanics/Latino are more likely to nap on the weekday while other researchers have suggested that naps are more likely to happen on the weekends [39]. In other studies, Black and Hispanics slept an hour less than Whites [40–47].

Outside of epidemiological data, community-based findings with similar trends further validate the notion that sleep health differences do exist across the lifespan. In a cross-sectional community study of children, 39.1% of Black children reported poorer sleep duration and more naps compared to 4.9% White children. To explain this twin phenomenon of sleep deprivation and napping, it is likely that Black children who are sleep deprived try to catch up on lost sleep, through napping, and extended sleep duration over the weekend than weekday [44, 48, 49]. Further stratification by sex also shows race-sex differences in sleep duration. In a community study in Chicago, Illinois White men (6.7 hours) have the highest average sleep (actigraphy), then White women (6.1 hours), then Black women (5.9 hours), and then Black men (5.1 hours) [36], a trend observed across other studies [47, 50–53]. The burden of short sleep duration and sleep deprivation, among racial ethnic minorities, is consequential as they are linked with several chronic health conditions [46, 54–58].

Sleep Disorders

It is estimated that 50–70 million people have a sleep disorder, with obstructive sleep apnea (OSA) and insomnia being the two most prevalent in the United States. OSA is a sleep breathing disorder characterized by partial or complete blockage of the upper airway, resulting in reflexive awakenings and transient cessation in breathing patterns (apneas and hypopneas). Apnea and hypopnea events cause oxygen desaturation and physiological stress, thus affecting key homeostatic physiological processes. Key OSA symptoms include: sleep-related pauses in respiration, arousals, unrefreshing sleep, snoring, restlessness, poor concentration, fatigue, and excessive daytime sleepiness [34]. OSA is considered one of the most common sleep disorders, among middle and older aged adults, affecting around 24% and 49.7% of the US population [52]. Obesity, large neck size, instability in respiratory control system, and craniofacial structures are key OSA risk factors. OSA is associated with cardiovascular disease, cardiometabolic conditions, cerebrovascular, low and worsen cognitive performance, and dementia [34, 59, 60].

The burden of OSA risk and disease is high among racial-ethnic minorities (among pediatric and adult populations), specifically Blacks (See Table 3.1). Blacks children aged 2–18 were more likely to experience sleep-disordered breathing (SDB), even after controlling for specific variables, obesity, respiratory problems, smoking, and neighborhood of residence. Even racial and ethnic parents have

reported that their child snores more than non-ethnic parents. Other estimates indicate that Black children are 4–6 times more likely to have OSA, Hispanic/Latinx children have a greater severity, Native American children are 1.7 times more likely to have moderate to severe OSA, and Asian Americans have similar or lower OSA prevalence compared to White children [59]. In an adult population using data from the Jackson Heart sleep study (n = 852), approximately 24% of the sample had moderate to severe OSA based on apnea-hypopnea index (a measure of severity), but only 5% had a diagnosis, indicating that the overwhelming majority of participants were undiagnosed (95%). Black men had a higher prevalence of OSA compared with Black women [61]. The foregoing evidence suggests that even when Blacks experience OSA symptoms, they were less likely to be diagnosed and treated. Similar trends are observed among Latinx and Asian popualtions [34], with 49.4% of Latinos and 43.1% of Asians reporting significant snoring, a major OSA symptom and risk factor. Although population estimates are high, community-level estimates indicate even higher burden among racial/ethnic minorities. In a study conducted in primary care community-based clinics in Brooklyn NY, almost half of Black patients (45%) reported debilitating snoring and about one-third reported excessive daytime sleepiness (33%) and difficulty maintaining sleep, a sign of insomnia (34%) [6]. However, for Latinx population being overweight/obese was the strongest marker and predictor of OSA risk, while for Asians craniofacial features and not body adiposity was most predictive of OSA risk [62]. The heterogeneity in OSA risk across racial/ethnic groups has proved difficult for adequate screening, assessment, and treatment, often leading to high rates of untreated individuals. The consequence of untreated OSA has proved consequential, especially among Blacks, as it is linked to elevated, uncontrolled, and resistant blood pressure and stroke [34, 59, 60].

OSA burden and disparity are not just observed and confined to differential estimates of the disease but also rooted in the uneven distribution of upstream and downstream consequences of OSA, as well as the lack of system level infrastructures to attenuate or buffer these burdens. Adherence to OSA treatments is a major problem among racial and ethnic minority groups as Blacks have one of the poorest OSA treatment (positive airway pressure [PAP]) adherence rates [63]. Poor treatment adherence is credited more to system-level barriers in the healthcare system such as poor insurance coverage, under-resourced sleep clinics in predominantly low-income and minority neighborhoods, and the limited amount of board-certified minority clinicians and providers.

Insomnia is another prevalent sleep disorder among racial/ethnic minorities. Although the prevalence of an insomnia diagnosis is mixed among minority groups, the prevalence of insomnia symptoms such as involuntary early morning awakenings, difficulty falling asleep, and issues with staying asleep are high. For racial and ethnic groups, insomnia is one of the most common sleep complaints and disorders, with approximately 30% reporting at least one insomnia symptom, 5–10% meeting threshold for an insomnia disorder, and approximately 6% with an actual diagnosis [51, 64, 65]. Insomnia's nocturnal symptoms and daytime consequences include lack of energy, difficulty concentrating, fatigue, tiredness, irritability, and

moodiness. Insomnia disorder increases the risk of stress, anxiety, depression, and decreased quality of life.

Several population studies show that Blacks are more likely to be affected by insomnia symptoms compared to other racial/ethnic groups. In a US National Institute of Health (NIH) study with 825 Black Americans (both men and women), 1 in 5 participants had insomnia and 6.7% an insomnia diagnosis. Another study demonstrated that Blacks reported greater nighttime insomnia relative to their other racial/ethnic counterparts.

Chronotype and Circadian Rhythms

Observed differences in circadian rhythms, chronotype, and sleep timing (irregular sleep time between weekdays and weekends such as social jetlag) among racial/ethnic minorities relative to Whites is well-documented (See Table 3.1). Circadian rhythms is the 24-hour internal clock that regulates the scheduling of important bio-behavioral activities such as eating, metabolizing food, sleep and rest, and when a person is most active or stressed. The circadian master clock, the superchiasmatic nucleus, coordinates, and synchronizes with peripheral clocks in the body (i.e., heart, cells) to ensure all biological and functional processes are aligned, synchronized, and working optimally. Circadian rhythms are influenced by exogenous cues and stimuli such as light, darkness, and sound that help punctuate the day and signal shifts that help the body regulate itself. However, desynchronized and chronic exposure to these cues and stimuli can result in circadian dysregulation and possible misalignment. Circadian misalignment occurs when an individual's central and peripheral biological clocks become misaligned from their daily behavioral clock, such as the time an individual the routine sleep, meal, and activity [45, 57, 66–69]. The desynchrony of exogenous, endogenous, and behavioral clocks can occur as early as childhood and is linked to poor emotion regulation and obesity across the lifespan [57, 70].

Chronotype among minorities Persons with evening chronotype are less likely to have regular exercise and more likely to partake in unhealthy diets and lifestyle choices that increase their cardiovascular risk. In a study made up of 61.5% of racially and ethnically diverse women in the United States (N = 506) greater morningness was associated with a more favorable cardiovascular profile which included BMI, blood pressure, cholesterol, and glucose levels compared to their white counterparts. Conversely, compared to the morning chronotype, evening persons had a greater than two-fold higher odds of having a poor cardiovascular profile and sleep duration of less than 7 hours per night [71]. Among adults in the Southern region of the United States, obesity was significantly associated with evening chronotype in whites, but not blacks even after adjusting for important covariates like shift work, physical activity, and sleep duration which could suggest the need for more pointed

research exploring the multidimensional nature of racial and ethnic chronotypes [71].

Circadian rhythms among minorities Several studies show that Blacks have shorter free-running circadian periods (tau) than Whites (24.07 hours vs 24.33 hours). Shorter free-running circadian periods make it more difficult to adjust to night-shift work and delayed (daytime) sleep schedule [67–69]. The health consequences of shifts in circadian rhythms can be grave, as studies indicate that Black night shift workers are more likely to have elevated blood pressure and hypertensive compared to Black day workers. In a community sample of Blacks in New York City, Black shift workers had 35% increased odds of having hypertension among Blacks [OR = 1.35, CI: 1.06–1.72. $P < 0.05$], compared to their White counterparts. Circadian rhythm disruption among Blacks who work non-traditional hours leads to sleep deprivation and shorter sleep duration and 80% increased cardiovascular disease risk such as hypertension [OR = 1.81, CI: 1.29–2.54, $P < 0.01$] [67]. Blacks who have shorter circadian periods and live closer to the equator with longer exposure to sunlight were less likely to have disrupted circadian rhythms.

Social jetlag among minorities A prevalent phenomenon that may impact sleep habits is the concept of "social jetlag". Social jetlag occurs when an individual's weekday and weekend sleep time is significantly different from their body's endogenous circadian clock. This results in poorer sleep quality, sleep time in deep sleep, and may result in other adverse functional and health outcomes. Disruptions in sleep timing can have ripple effects on the timing of other key social and biological activities such as eating. It is highly likely that disruptions in sleep timing due to social jetlag may also result in eating jet lag. Eating jetlag occurs when an individual's meal timing is misaligned with their endogenous metabolic circadian clock. Combined, irregular sleep, physical activity, and mealtimes are key contributors to circadian misalignment and has been linked to adverse functional and health outcomes. Social jetlag can have severe and adverse health consequences. For example, the New Hoorn study cohort ($n = 1585$) investigated the association between social jet lag, metabolic syndrome, cardiovascular health and found that individuals younger than 61 years of age who reported social jetlag (1–2 hours) had approximately a two-fold greater risk of metabolic syndrome and prediabetes/diabetes compared to their counterparts who reported less than 1 hour of social jetlag [72].

Sleep Architecture

Sleep architecture represents the cyclical pattern of sleep as it shifts between the different sleep stages, non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. An individual's sleep architecture is made up of sleep cycles and stages marked by unique neurological, autonomic, and physiological signals that correspond to the stage of sleep or wake an individual is experiencing [61, 73]. Each

full cycle of NREM and REM sleep lasts about 90 to 120 minutes. NREM sleep is characterized by 3 stages of sleep (N1, N2, and N3). Stage 1 of NREM is the lightest form of sleep marked by low amplitude alpha brain waves, Stage 2 is marked by sleep spindles and K complexes, and Stage 3 is marked by higher amplitude delta waves signifying deeper more restorative sleep. A normal pattern of sleep cycles (as shown in the hypnogram) includes a greater portion of time spent in Stages 2 and 3 sleep at the beginning of the night and more REM sleep at the second half of the night, with a few possible brief awakenings scattered throughout the sleep stages.

Racial/ethnic differences, in the quantity and quality of sleep architecture, are well documented, where Blacks tend to get more light sleep and less deep sleep compared to Whites (See Table 3.1) [48]. In the Outcomes of Sleep Disorders in Older Men (Mr OS Sleep) Study with Black, Asian American, Hispanic and White men ($n = 2823$), Black men relative to other races/ethnicities had the lowest percentage of Stage 1 non-REM sleep (6.59%) and slow-wave sleep Stage 3 sleep (7.99%). However, Blacks had the highest percentage of Stage 2 non-REM sleep (64.79%) and REM sleep (20.71%) [74]. Overall, Blacks spend less time in slow-wave sleep and spend greater time in REM relative to Whites. Therefore, it is likely that sleep-deprived Blacks are more likely to experience daytime sleepiness and physical fatigue.

Sleep Efficiency

Sleep efficiency is another sleep health parameter that racial/ethnic differences can be observed. Overall, sleep efficiency captures how much sleep an individual actually experiences and is predicated on several sleep characteristics, such as sleep latency and wake after sleep onset. In a population-based study, Black men had lower sleep efficiency (79.7%), due to longer sleep latency (29.4 minutes) and greater WASO (90.4%) relative to Whites, Hispanic/Latinx, and Asian Americans [74]. These results suggest that Blacks took a longer time to fall asleep, had less efficient sleep (meaning that they spent less time sleeping while in bed), and had the greatest levels of awakenings after sleep was initiated.

Sleep Quality and Sleepiness

Racial/ethnic minorities have a higher burden of daytime sleepiness and poor sleep quality compared to Whites (See Table 3.1). In the Multi-Ethnic Study of Atherosclerosis (MESA) study, Blacks had the highest rates of excessive daytime sleepiness (using the Epworth Sleepiness Scale [ESS] score > 12) at 13.1%, compared to Hispanic/Latinx at 9.2%, Whites at 8.0%, and Chinese at 7.5% [75]. However, Whites reported highest rate of being sleepy for more than 5 days of the month (18.6%) compared to the other racial/ethnic groups (Black = 14.4%, Hispanic/

Latinx = 16.9%, and Chinese = 12.3%). Compared to Whites, Blacks, Hispanic/Latinx, and Chinese Americans had lower odds of excessive sleepiness ≤5 days/month, after adjusting for demographic factors. However, after adjusting for physical health and psychosocial variables, differences in sleepiness between Whites and Chinese Americans decreased suggesting that these factors play a crucial role in the estimates on the amount of sleepy days among Chinese Americans. Relative to Whites, Black Americans had the highest odds (and only, as Hispanic/Latinx and Chinese were not significant) of excessive daytime sleepiness (ESS scale >12) (ORs range across 4 adjusted models 1.43–1.74, adjusting for sociodemographic, psychosocial, physical health, and sleep duration and disorders). The greatest attenuation in the differential estimates of daytime sleepiness between Blacks and Whites was observed when controlling for physical health and sleep variables, but not for psychosocial factors.

Causes of Poor Sleep Health

The root cause of sleep health disparities among racial/ethnic minorities is multifarious and complex, as several biological, psychosocial, and environmental factors may explain the burden of poor sleep health.

Biological Causes

Empirical studies highlight several biological causes of racial/ethnic differences on sleep health parameters. Biological explanations for sleep health disparities include genetic, circadian, and anthropometric. Although genetic causes of differential sleep health estimates are intricate and inconclusive, initial evidence from large genetic studies indicate a possible ancestry link. For example, in a study that investigated 1698 ancestry genetic markers, individuals with higher percentage of African ancestry had lower percentage of slow-wave sleep SWS and explained 11% of the variability in slow-wave sleep, a marker of sleep depth [76]. As indicated above, Blacks or individual with African Ancestry have shortened free-running circadian period (tau) compared with their White counterparts [68]. Anthropometric causes may also explain differential estimates of sleep health outcomes. In a study comparing the link between craniofacial features and risk for sleep apnea among Asian and White Americans, neck circumference, body mass index, mallampati score (MS) (measurement of tongue and mouth during a breath hold at end-tidal inspiration with the mouth wide open and tongue fully protruded), thyromental distance (TMD), and thyromental angle (TMA) (angle between the soft tissue of backside of neck, the soft tissue mentum, and the thyroid) were the best predictors of OSA risk and severity [77]. Asians had different MS, TMD and TMA compared to Whites which was associated with greater sleep apnea severity and had higher MS, smaller

TMD, and larger TMA. In another study, obesity explained sleep apnea risk among Whites, while skeletal restriction explained sleep apnea risk among Chinese. Despite this difference, the ratio between obesity to craniofacial bone size, a determinant of upper airway volume and OSA risk, was not statistically different between Chinese and Whites [78].

Psychosocial Causes

There are several psychosocial factors that might explain sleep health disparities among racial/ethnic minorities. These factors include but are not limited to social stressors, beliefs, and attitudes and behaviors.

Social stressors Several researchers suggest that racial inequalities and social inequities developed through racial segregation, food desserts, lack of resources, educational attainment, employment status, and limited to no access to health care system care can be linked to psychological distress, anxiety, depression and poor sleep and unhealthy sleep behaviors [46, 79, 80].

In a sample of 4863 Black adults, psychosocial stressors such as perceived stress, major life events stress, and weekly stress were associated with short sleep duration and poorer sleep quality. The effects of weekly stress on sleep duration was most pronounced among younger (<60 hears old) and college-educated Blacks [81]. Similar trends can be observed for the Latinx population, where depressive symptoms, employment status, and low education level were independently associated with short sleep duration, while unemployment, low household income, and low level of education were independently associated with long sleep [82]. In a study that explored the influence of perceived racial discrimination and the risk of insomnia on middle-age elderly Black women ($N = 26{,}139$), participants with higher perceived levels of discrimination had higher insomnia symptoms and shorter sleep duration (<7 hours) [83]. While in another study, economic disadvantage and poor physical and mental health were statistically were associated with insomnia among older Blacks ($N = 398$) in Southern Los Angeles [35].

Beliefs and attitudes Racial ethnic minorities' beliefs and attitudes about sleep and sleep health play crucial roles in the amount of sleep an individual receives and the quality of their sleep. The association beliefs and attitudes have on sleep outcomes is likely to be indirect and reflects a mediated association between inadequate sleep health literacy, unhealthy sleep behavior, and poor sleep health outcomes. Individuals may not know or appreciate the importance of sleep and how it impacts their health and functional outcomes. For example, some racial/ethnic minorities have considered deep habitual snoring or snoring as relatively good sleep and are unaware that it may portend something more ominous such as a sleep breathing disorder like sleep apnea. In a study of community-dwelling Black men, participants with elevated and high risk for OSA were more likely to report false and maladaptive beliefs

about sleep [84]. In another study, Blacks reported using napping and consuming caffeine to cope with sleep deprivation and sleepiness. In the same study, participants reported using electronic devices (such as TV and phone) to blunt racing and ruminative thoughts that prevented them from falling asleep [85].

Behaviors There are a number of behaviors that can affect differential sleep estimates across racial/ethnic groups. These include but are not limited to mental health, diet, and physical activity. For example, studies have shown that Blacks with elevated emotional distress are more likely to report short or long sleep durations [86]. In a nationally representative study, insufficient sleep (<7 hours) was associated with unhealthy diets, suggesting a potential bi-directional relationship where poor sleep leads to poor diet and food choices and poor food choices, such as night eating and consumption of high-calorie foods close to bedtime may lead to later bedtime, late sleep onset, and disrupted sleep [87]. Other studies have found that Blacks and Whites respond differently to food stimulants like caffeine when they found that Blacks who consume caffeinated drinks were more likely to have disrupted sleep compared to Whites [50]. Physical activity/exercise is another behavior that might cause differential sleep health estimates between racial/ethnic groups and Whites. In a sample of 246 Black adolescents, physical activity protected against short sleep duration [88]. Specifically, race and sleep duration appeared to be only significant at lower levels of physical activity and Black adolescents who reported shorter sleep durations had lower physical activity.

Physical and Built Environment Causes

There is growing evidence that environment, physical and built, can affect sleep health outcomes. Patterns of Insufficient sleep is more prevalent in poor urban and rural settings relative to their more affluent counterparts [89]. These findings highlight that geographical effect on sleep health outcomes may traverse race/ethnicity, as majority of the region is White, although the amount of Black (4%), Asian (26%), and Hispanic (37%) residents have been increasing, according to a 2019 Pew Trust research poll. Outside of geographic patterns of and effects on sleep health outcomes, more granular evidence highlight the contribution of noxious noise, light and temperature have on sleep health outcomes, as well as physical environment of an individual's community influences their sleep. For example, social cohesion, safety, light, traffic, air quality and pollution, noise, greenspace, and neighborhood cohesion/disorder and walkability may impact sleep health outcomes. Data shows that racial/ethnic minorities might be particularly vulnerable to the effects of environmental factors on sleep health outcomes.

Of the environmental factors listed above, the role of light on sleep has the most robust evidence to date explaining racial/ethnic differences in sleep health outcomes. The proliferation and exposure to artificial light presents the clearest and

most present danger to sleep health. Artificial light (ALAN) during the day and mostly at night is harmful, and evidence points the unfortunate burden and vulnerability among racial/ethnic minorities [90]. Blacks and Hispanics when exposed to ALAN 2 times greater than Whites [56]. Dominant artificial light exposure from in-house sources such as laptops, individual's computer, cellphones, televisions, and outside sources including street lights are more likely to disrupt an individual's natural sleep-wake cycles causing circadian misalignment and sleep disruption.

Noise levels from several sources such as train, industrial activity, traffic, nocturnal noise can have deleterious effects on sleep health outcomes such as sleep disturbances, daytime sleepiness, irritated, frustrated, annoyance, inconsistent mood changes, and adverse to long-term effects on cardiometabolic outcomes. Excessive noise pollution may trigger stress hormones that can increase blood pressure, heart rate during sleep times, and autonomic arousals thus leading to microarousal that lead to shallow, fragmented, and unrestorative sleep. A study funded by Robert Wood Johnson and National Cancer Institute found that neighborhoods with predominantly Asians, Blacks, and Hispanics residents had higher levels of noxious noise levels during the day and at night (approximately 4 decibels higher on average) compared to neighborhoods without racial and ethnic groups [65].

Health Consequences of Poor Sleep Health

The third set of evidence to establish a health disparity is the higher burden of downstream consequences as a result of poor sleep health experienced by racial/ethnic minorities. Racial/ethnic minorities as at significantly higher risk for a host of adverse functional and health outcomes, such as cardiovascular disease, cardiometabolic conditions, and poor brain health (mental health and dementia).

First, poor sleep health, which includes sleep deprivation, shorter sleep duration, sleep disorders (sleep apnea and insomnia), and poor sleep quality, is directly linked to increased cardiovascular risks such as heart disease, high blood pressure, stroke, diabetes, and cardiovascular health and disease, among racial/ethnic minorities [36, 37, 86, 91–94]. For example, in the CARDIA study (n = 578; ages 33–45), shorter sleep duration predicted hypertension (OR 1.37, 95% CI: 1.05, 1.78) [95]. The direct association between poor sleep and adverse health outcomes among racial/ethnic minorities is due to high prevalence of sleep deprivation, where they are twice as likely to sleep less than their white counterparts.

Second, poor sleep health parameters are indirectly linked to adverse health outcomes among racial/ethnic minorities. Evidence of these indirect associations include the mediated role of shift work, where racial/ethnic minorities working night shift are at increased risk for circadian misalignment and cardiometabolic disease and poor mental health outcomes [25, 96–99]. The indirect association between sleep and adverse health outcomes is significant and consequential because Blacks and Latina/os are more likely to work non-traditional work shifts compared

to their White counterparts. Blacks who work night shifts are at greater risk and burden for hypertension compared to Blacks that work day shifts, whereas, for Whites no differences between day and night shift work were observed [50]. These racial/ethnic differences in shift work may partly explain the burden of hypertension in Blacks, as night shift workers have lower blood pressure dipping at night than day shift workers at the same time causing a prolonged elevated blood pressure. Prolonged elevated blood pressure can cause hypertension, resistant hypertension, and elevated risk for stroke, all health conditions highly prevalent among Blacks [53, 98, 100, 101].

The indirect and mediated relationships between sleep and adverse health outcomes are not solely due to social determinants of health or psychosocial factors but also may be engendered by biological, physiological, and anatomical factors [40]. Individuals who report insufficient sleep over a period of time have a higher caloric intake (+30% of daily caloric requirement), compared to Whites. Insufficient sleep and sleep deprivation may induce poor eating habits, thus increasing the higher caloric intake of carbohydrates, snacks, unhealthy foods, age, gender, and BMI, which can promote weight gain [102]. A meta-analysis of 72 studies found that in restricted sleep short and long sleep durations were associated with cardiovascular inflammatory markers such as: C-reactive protein and interleukin (IL)-6, factors linked with cardio-metabolic conditions (obesity and type 2 diabetes), neurodegenerative and pulmonary disease [79].

Several studies note significant brain health consequences – cognitive decline, cognition impairment, and neurodegenerative disease like dementia –as a result of poor sleep health among racial/ethnic groups. For example, excessive daytime sleepiness is associated with cognitive decline, impaired cognition, mood, executive decisions, minimal attention span, memory and emotionally memory, and inflammation of the brain [9, 10, 56–62]. Race stratified analyses indicate that the associations between sleep health parameters (notably daytime sleepiness, short sleep duration, and long sleep duration) and cognitive impairment/decline are most pronounced in Blacks and Hispanics compared to other racial/ethnic groups [43, 47].

In one population-based study (n = 28,756), the majority of participants with extreme sleep deprivation (less than 4 hours or more than 10 hours per night) experienced greater cognitive decline than individuals with at least 7 hours of sleep per night, [62] with racial ethnic minorities appearing to be most affected. In another study with middle-age adults, inconsistent sleep time had a negative impact on cognitive functioning as individuals showed clinically significant signs of cognitive decline after 3 weeks [103]. The adverse effects of poor sleep can have long-term effects on cognition. In a sample of Japanese-Americans, individuals with high levels of daytime sleepiness had a greater odds of dementia and cognitive decline in a three-year follow-up [104]. Findings from these studies highlight that two possibilities. First, poor sleep health may be a risk factor for acute and chronic cognitive decline. Second, sleep may serve as an early sign of cognitive decline, which may portend the onset of dementia.

Conclusion

The aim of this chapter was to describe extant estimates of poor sleep health among racial/ethnic minorities, multilevel determinants of sleep health disparities among racial/ethnic minorities, including biological, environmental, and psychosocial factors, and associated downstream health outcomes among racial/ethnic minorities. Over the past decades, important efforts have been made by health disparities clinicians and researchers to raise awareness about new approaches to tackle disparities in sleep health. However, data highlighted in this book chapter indicates that there is still a long road to travel until we arrive at sleep health equity in the United States [44, 49, 57, 59, 60, 63, 83, 105–108]. Building upon the sleep health framework, we suggest that one of the first crucial steps in addressing racial/ethnic disparities in sleep health is to define the concept of sleep health equity that we conceptualized as the equal opportunity to experience and obtain healthy sleep regardless of age, sex, race/ethnicity, geographical location, and socioeconomic status to obtain satisfactory sleep that promotes physical and mental well-being [28]. We also argue that achieving sleep health equity requires a multi-level and multisystem approach that includes patients, providers, payers, and the entire healthcare ecosystem. To achieve sleep health equity involves the following five steps and initiatives [28]:

1. We encourage implementation of sleep health literacy programs for all ages – from early screening and treatment for sleep disorders starting in preschool, elementary schools, high schools, and university. This will provide sleep health literacy modules, workshops, webinars, throughout websites, social media outlets, and mobile apps.
2. This awareness and sleep health access for ethnic and racial minorities could be achieved through the creation and multilingual sleep centers in vulnerable communities. Such multi-ethnic and multi-lingual initiatives could also be replicated in additional health centers located in vulnerable communities.
3. Culturally tailored behavioral sleep health interventions may increase adherence to physician's recommendations among racial and ethnic minorities. For instance, this could be achieved through required, culturally sensitive training in sleep medicine programs where physicians are better equipped in administering sleep health medicine and interventions for vulnerable populations.
4. We need training programs across all educational levels from high school to university (Ex: the New York University's PRIDE and COMRADE programs), which aims to increase diversity in the sleep health medicine workforce.
5. Public health policies to address and reduce the burden of environmental (ex: noise and light) exposures that are underpinning poor sleep health among racial/ethnic minorities are strongly encouraged. This is very important because Blacks and other minorities experience a higher rate of environmental risk living in disfranchised neighborhoods and communities.

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