Published in final edited form as:

Sleep Health. 2017 October ; 3(5): 401–415. doi:10.1016/j.sleh.2017.08.001.

Determinants of Racial/Ethnic Disparities in Disordered Sleep and Obesity

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Abstract

Racial/ethnic minorities experience a disproportionate risk of both suboptimal sleep and obesity, and the relationship between sleep and obesity may differ by race/ethnicity for modifiable and non-modifiable reasons. Since many non-White populations have historically and continue to largely live in disadvantaged, obesogenic physical and social environments, these greater inopportune exposures likely adversely affect sleep, resulting in physiological dysregulation. Physiological dysregulation may, in turn, lead to increased obesity risk and subsequent health consequences, which is likely more influential than potential genetic differences in race - a social construct. The purpose of this article is to describe potential environmental, genetic, and epigenetic determinants of racial/ethnic differences in the sleep-obesity relationship and to review current epidemiological findings regarding either racial/ethnic minority specific estimates of the association or disparities in the relationship. Using the socioecological framework as a conceptual model, I describe sleep and obesity as socially patterned and embedded in modifiable physical and social contexts with common causes that are influenced by upstream social conditions. I also provide examples of sleep and obesity-related studies that correspond with either the downstream, intermediate, and upstream factors that likely contribute to commonly-observed racial/ethnic disparities in the sleep-obesity relationship. The review concludes with broad recommendations for 1) advancing research methodology for epidemiological studies of disparities in the link between sleep and obesity, 2) future research topics, as well as 3) several broad policies and structures needed to address racial/ethnic disparities in sleep health and obesity.

Keywords

Sleep disorders; Obesity; Race; Health Status Disparities

PUBLIC HEALTH IMPORTANCE OF SLEEP

Optimal sleep is of tremendous public health importance. Out of the roughly 320 million US citizens, approximately 50-70 million US adults have sleep or wakefulness disorders like sleep apnea and insomnia¹. Although a matter of debate, sleep duration appears to be decreasing with Americans reporting, on average, 6.1 hours of sleep per day²⁻⁴, and

lack of sleep has been shown to cost the US economy approximately \$411 billion per year⁵. Sixty-million prescriptions for sleeping pills are written per year, and over 300,000 (6,400 fatal) accidents occur annually, which are largely attributed to drowsy driving⁶. Daytime sleepiness and difficulty falling asleep affect nearly 35 to 40% of US adults per year⁷ and contribute to poor performance, mood disorders like anxiety and depression, along with a host of poor health outcomes, including obesity, hypertension, type 2 diabetes, cardiovascular disease, and premature mortality⁸. The Institute of Medicine has identified sleep as an unmet public health need, and the Department of Health and Human Services has established a Healthy People 2020 goals to, for instance, increase the proportion of Americans getting the recommended amount of sleep while acknowledging that all human sleep needs likely vary between individuals¹. Currently, 35% of American adults report habitual short sleep and 8% report being long sleepers⁹.

Regarding physiological stages of sleep, a person typically falls asleep in a light stage and, over approximately 90-minute repetitive cycles, the individual progresses into deeper periods of non-Rapid Eye Movement (REM) sleep, which comprises, on average, 75% of the sleeping period and REM sleep comprises 25%¹⁰. Different stages of sleep, as measured by frequency and amplitude of neurophysiological signals from polysomnography, is associated with particular health benefits. For instance, slow wave or deep sleep (denoted as “N3”) is associated with physiological restoration^{11,12}. During this stage, cellular and tissue growth and repair occurs, growth and other metabolically-active hormones are released, blood pressure normally drops, and breathing slows down. REM sleep is associated with cognitive restoration, which is important for learning, memory consolidation, and emotion regulation¹³. Sleep duration recommendations differ across the lifecycle and since different stages are associated with a particular health benefit, it is recommended by the National Sleep Foundation that adults get 7 to 9 hours of quality, uninterrupted sleep in order for sleep to be fully restorative¹⁴. A more recent consensus statement from the American Academy of Sleep Medicine and Sleep Research Society have recommended that adults get at least 7 hours of sleep on a habitual basis without the upper limit of 9 hours because sleep deprivation is common and sleep-deprived individuals should prioritize “paying down their sleep debt” over meeting the established sleep recommendations¹⁵.

PUBLIC HEALTH IMPORTANCE OF OBESITY

Second to cigarette smoking, obesity is considered the 2nd leading cause of preventable death and annually costs the US economy \$147 billion¹⁶. Thirty-five percent of US men and 40% of women are classified as obese¹⁷, which is an established risk factor for various potentially devastating chronic diseases. For instance, it is estimated that 75% of hypertension incidence is related to obesity^{18,19}. Obesity is also associated with an increased risk of type 2 diabetes²⁰, cardiovascular disease^{21,22}, and premature mortality²³. The relationship between sleep and obesity has been relatively understudied, but prior studies have revealed a likely bidirectional relationship where sleep restriction increases risk of obesity and, in turn, obesity increases risk of disrupted sleep (most notably as a consequence of snoring and sleep apnea)²⁴.

THE RELATIONSHIP BETWEEN SLEEP AND OBESITY

Suggesting a shared influence or interconnectedness, several of the same endogenous (e.g. cortisol) and exogenous (e.g. nutrition, physical activity, psychosocial stress) factors independently affect both sleep and obesity²⁵, and many organ systems (e.g. endocrine, nervous, reproductive) are affected by both sleep and obesity. In fact, the prevalence of both suboptimal sleep and obesity have increased over the past several decades²⁶, and have common risk factors (e.g. stress) as well as physical and mental health (e.g. depression) consequences. Suboptimal sleep characteristics and sleep disorders (independent of body mass index) are associated with hypertension²⁷, diabetes²⁵, cardiovascular disease²⁸, and mortality^{29,30}.

Regarding age, associations between sleep and obesity have been consistently stronger in children compared to adults²³. Children and adolescents may be more vulnerable to the effects of insufficient sleep as it is important for brain development, and the effect of sleep on weight gain may be altered over time in that short sleepers may not continue to gain weight in a linear fashion.

BIOLOGICAL, BEHAVIORAL, AND SOCIAL PATHWAYS LINKING SLEEP AND OBESITY

There are numerous plausible biological, behavioral, and social pathways by which sleep may directly or indirectly affect obesity. In general, weight gain and obesity occur because of an imbalance of energy intake, energy expenditure, and energy storage over time. Humans expend energy through thermogenesis (~10%) to absorb and metabolize food, physical activity (~15%), and resting metabolic rate (RMR) (75%), which supports bodily functions like brain activity, breathing, circulation, and digestion³¹. A circadian rhythm for RMR exists which also varies by stage of sleep³². RMR typically decreases and is lowest at night during nocturnal sleep, and it typically rises toward the morning until it peaks in the afternoon. Insufficient sleep could lead to disrupted synchrony between sleep/wake cycles over time that may lead to a cascade of negative effects on, for example, RMR, glucose metabolism, neuroendocrine responses, and behavioral changes that increase food consumption and decrease energy expenditure, thereby eventually leading to obesity³³.

Furthermore, the hypothalamus of the endocrine system regulates the pineal gland that synthesizes and secretes hormones like melatonin and the pituitary gland secretes many other sleep- and metabolism-relevant hormones³⁴. The hypothalamic-pituitary axis regulates hormones important for both sleep and obesity. For instance, stress is believed to negatively affect sleep duration and quality as well as obesity risk, and cortisol (a well-established stress hormone) follows a circadian rhythm that is a melatonin antagonist and is associated with the accumulation of visceral adiposity³⁵. Sleep restriction has also been shown to decrease insulin sensitivity, impair glucose tolerance, and upregulate the orexigenic hormone ghrelin found in the stomach, which is associated with increased hunger (see Figure 1)^{10,36}. Not getting enough sleep is associated with the down regulation of leptin, a hormone secreted from adipose tissue, which is associated with satiety or being satisfied after a meal. These metabolically-active appetite-regulating hormones may act in

concrete to increase risk of weight gain and eventual obesity through overeating^{37,38}. Another hypothesis relates to activation of reward areas of the brain during partial sleep restriction that may lead to a greater propensity to overeat^{39,40}.

Behavioral mechanisms linking sleep to obesity include an individual who gets less sleep having more time available to consume food⁴¹. A person habitually getting less sleep is also more likely to be fatigued and, therefore, less likely to engage in physical activity, which could contribute to an increased risk of obesity due to caloric surplus. Multimedia use like television watching can contribute to sleep deprivation, increased sedentary behavior, and increased caloric intake through, for example, exposure to advertisements^{42,43}. Stress is also generally associated with consuming more calories overall as well as consuming a less healthy dietary pattern⁴⁴. Compared to well-rested counterparts, people with insufficient sleep have been shown to consume approximately 300 more calories a day with most of the additional calories coming from carbohydrates or high-fat foods⁴⁵. Average weight gain is 0.5 to 1 kg per year⁴⁶, and this incremental accumulation of weight over time can eventually lead to obesity. Individuals may eat more than needed to cover the energy demands related to staying awake longer. Timing of food consumption in relation to sleep may also be independently important for body weight regulation. Studies have shown that daytime eating is associated with lower triglycerides, cholesterol, and delayed or nighttime consumption is associated with decreased fat oxidation and increased cholesterol⁴⁷.

With regard to social pathways, we all live in a 24-hour society that – with the advent of artificial electric light and advancing technology – can contribute to less time spent sleeping⁴⁸. Some individuals or groups of people may have less opportunity to acquire the recommended amounts of sleep for a wide range of reasons. For instance, individuals of lower socioeconomic status may live in physical and social environments that are more likely increase obesity risk through infringing on sleep through noise and light pollution, working rotating-night shifts or extended hours due to low wages, and stress induced by the threat or reality of violence, making the affected individuals more vulnerable or susceptible to insufficient sleep duration and inadequate sleep quality⁴⁹⁻⁵³.

RACIAL/ETHNIC DISPARITIES IN SLEEP AND COMMON SLEEP DISORDERS

This section summarizes racial/ethnic disparities in sleep duration, quality, and the two most common sleep disorders, obstructive sleep apnea and insomnia. Compared to Whites, African-Americans or Blacks are nearly twice as likely to report short sleep duration with Black men being the most likely to be short sleepers⁵⁴⁻⁵⁷. On average, Blacks sleep approximately 1-hour less than Whites over a 24-hour period. Blacks are also over 60% more likely to report being long sleepers⁵⁵. Furthermore, there appear to be differences in sleep architecture by race. For example, Blacks have been consistently shown to experience less deep or slow wave sleep (stage N3) and more light sleep (stages N1 and N2)⁵⁸⁻⁶⁰. Blacks also tend to take longer to fall asleep (longer sleep latency) and have less efficient sleep or more time awake during the sleep period^{56,61,62}.

The prevalence of diagnosed obstructive sleep apnea (OSA) is particularly high among racial/ethnic minorities, and there is evidence that the rates of undiagnosed OSA are particularly high among racial/ethnic minorities (especially among Chinese) ⁶³. Among children, Black children have a 4-to-6 times higher prevalence of OSA than White children, which is partly attributed to being more likely to live in economically-deprived neighborhoods ⁶⁴. This exposure to sleep disorders earlier in life could lead to a greater cumulative burden of disease. Blacks overall also experience more severe health consequences in that this group is significantly more obese (women in particular) and have higher rates of hypertension at the time of OSA diagnosis, which may be due to the longer life-time burden from having sleep disorders earlier in the life course. Furthermore, there is a stronger association of increasing body mass index (BMI) and waist circumference with sleep-disordered breathing among Chinese individuals compared with Whites, Blacks, and Hispanics, which is not explained by sociodemographic characteristics, comorbidities, or lifestyle behaviors ⁶³. Therefore, some groups may be particularly susceptible to the adverse health effects of obesity.

There are inconsistent associations between insomnia and race/ethnicity in terms of which groups are most at risk ^{65,66}. Estimates may be biased due to methodological issues related to different definitions of insomnia, an overreliance on self reports, and the need for a healthcare professional's diagnosis, which is influenced by healthcare access and utilization that varies by race and ethnicity.

RACIAL/ETHNIC DISPARITIES IN OBESITY

Obesity prevalence is higher among Blacks and Latinos than Whites and Asians in the US, and the disparities are highest between Black and White women with approximately 57% of Black women being categorized as obese compared to 38% of White women (see Figure 2) ¹⁷. Figure 3, also using nationally representative data, illustrates that not everyone has been affected by the obesity epidemic and that there is socioeconomic variation in obesity trends within racial categories ⁶⁷. For example, only Black men with greater than a high school education have a consistently higher mean BMI than White men of any level of education across the study period while mean BMI for Black men with a less than high school education remained stable. Since sleep duration is generally believed to have decreased during this time period and because Black men generally have the highest prevalence of short sleep ²⁻⁴, this finding among Black men leads to important research questions related to sleep and socioeconomic status among Blacks.

RACIAL/ETHNIC DISPARITIES IN THE SLEEP-OBESITY RELATIONSHIP

While our understanding of the sleep-obesity relationship is limited, there is even less known about associations between sleep and obesity in non-White populations who are generally at higher risk for obesity. Krueger et. al. reviewed seven potential biological, behavioral, and socioenvironmental mechanisms that may explain obesity disparities, including: the thrifty gene hypothesis, developmental and epigenetics, diet and physical activity, sleep duration; screen time and sedentary behaviors, neighborhood context, and social networks ⁶⁸.

Previous epidemiological studies that have explicitly investigated racial/ethnic differences or included race-specific estimates of the association between various sleep measures and obesity are summarized in Table 1 for adults and Table 2 for children/adolescents. Like obesity, sleep can be investigated as an exposure, outcome, mediator, or moderator. Overall, there were 38 relevant epidemiological studies published from 1999 to July 2017 ^{24,63,68-112}. The clear majority were observational with a cross sectional design, 7 were prospective with an average study period of 4.4 (range: 2-8) years, and only 1 was experimental. Fifty-five percent of the studies were conducted among adults vs. children/adolescents, and sample sizes ranged from 69 to 30,133 participants. Although most studies in the general sleep-obesity literature are among White Americans or Europeans, 74% of these studies with racial/ethnic minorities included Black participants, 61% Latino, 32% Asian, and 24% other (e.g. American Indian, Native Hawaiian). Sixty-one percent of studies had White comparison groups and some included racial/ethnic minority groups alone or explicitly examined differences across subgroups (e.g. Mexican American vs. Puerto Rican) of the same racial groups. Measures of sleep characteristics were mainly based on self-report and most studies included BMI based on measurements vs. self-report.

This section describes prior literature that explicitly included racial/ethnic minorities or investigated racial differences in either sleep duration, sleep quality, daytime sleepiness, sleep disordered breathing (including OSA), or insomnia in relation to obesity risk among both adults and children/adolescents.

Sleep Duration and Obesity

Reviewing experimental and observational studies, Ranjarai et. al. found support for an association between inadequate sleep duration or impaired sleep quality and risk of cardiometabolic disease (including obesity), and concluded that suboptimal sleep may partially mediate cardiometabolic disease disparities ⁸¹. Among adults, directly examined racial differences in the relationship between sleep duration and obesity were mixed, but many have reported that short sleepers were more likely to be obese compared to those who get the recommended amount of sleep. Most studies reported racial differences in the sleep-obesity relationship ^{70,79,87,88,91,93}. For instance, short (5-6 hours) vs. recommended (7-8 hours) sleep was associated with a 17% increased odds for objectively-measured obesity (OR, 1.17 [95% CI, 1.00-1.38]; $P<0.05$) in adjusted analyses ⁷⁹, and racial/ethnic differences in patterns of risk varied with the relationship between very short sleep and obesity strongest among Blacks. Another study found that Black and Hispanic adult participants aged 18–39 years who reported sleeping 5 hours/night experienced a larger increase in BMI, visceral fat, and subcutaneous fat over five years compared to participants who slept 6-7 hours/night ¹¹³. Sleep duration was not associated with BMI or body fat in those 40 years. Most of these studies relied on BMI, but some included waist circumference as measures of abdominal obesity ^{71,91,94}. Some studies among adults, however, did not provide support for racial differences in the sleep-obesity relationship ^{71,83,94,113}.

Less is known about the sleep-obesity link among children and adolescents compared to adults, and race was often found to be an effect modifier of the relationship in these younger samples ^{73,80,82,86,90,96,97,100,102-104,106}. An experimental study among mainly racial/ethnic

minorities concluded that the promotion of household routines (particularly increasing sleep duration and reducing TV viewing) was associated with a decrease in BMI ¹¹⁴. In another study where racial differences were not reported due to 81% of the 785 participants being White, shorter sleep duration in 6th grade was independently associated with a greater likelihood of “overweight” (95 percentile) in 6th grade ¹⁰³. Shorter sleep duration in 3rd grade was also independently associated with “overweight” in 6th grade, independent of the child's weight status in 3rd grade. Several studies with racial/ethnic minority either adults or children that did not explicitly investigate the sleep-obesity link, generally found that sleep restriction or short sleep was associated with greater caloric intake ¹¹⁵⁻¹¹⁷, that sleep was a behavioral risk factor for obesity ^{100,104,118-120} and even compromised academic outcomes ¹²¹.

Sleep Quality/Disturbance and Obesity

The studies investigating impaired sleep quality/disturbance and obesity associations were conducted among adults and generally found positive associations among racial/ethnic minority groups ^{76,85,101,105}. For instance, a study of Black, young adult women residing in an urban setting found a significant positive association between the latency component of sleep quality and BMI despite a non-significant correlation between BMI and global sleep quality ⁸⁵. In another study of 1,515 Black metropolitan Atlanta residents, sleep disturbance was associated with a 48% increased odds of obesity compared to those without sleep disturbances, which was further modified by perceived stress ¹⁰¹. Obesity predicted waking up several times per night while other health conditions were associated with other poor sleep characteristics among 1,085 Mexican-American study participants where 12.6% reported trouble falling asleep, 30% woke up several times per night, 11.4 % had trouble staying asleep, 9.4% did not awake feeling rested, and 16.6% reported poor sleep quality ¹⁰⁵. Also, Yu et. al. found that sleep disturbance and stress eating mediated the relationship between depressive symptomatology and obesity ⁷⁶.

Daytime Sleepiness and Obesity

Daytime sleepiness effects multiple sleep disorders and is associated with impaired vigilance, greater fatigue as well as physical and mental health problems. Daytime sleepiness and obesity risk was also studied among racial/ethnic minorities ^{83,85,91,108}. One study found that among mostly Black women with extreme obesity, there was a significant positive relationship with BMI and overall fatigue severity, and BMI was positively correlated with six fatigue severity subscales ⁸⁵.

Obstructive Sleep Apnea/Disordered Breathing and Obesity

Most of the studies investigating the association between OSA and BMI reported significant racial/ethnic differences ^{63,78,91,93-95,99,122}, and some also included measures of waist circumference ^{63,78,99,122}. For instance, Chen et al. found that sleep disordered breathing (SDB) – a general term for breathing difficulties (ranging from snoring to OSA) experienced during sleep – was most prominent in Chinese after BMI adjustment ⁶³. In this same study, Blacks, Hispanics, and Chinese had higher odds of short sleep than Whites irrespective of BMI and Blacks with either normal weight or obesity had worse sleep quality than Whites. A review conducted by Sutherland et al. concluded that Asian OSA populations primarily

displayed features of craniofacial skeletal restriction, Blacks displayed more obesity and enlarged upper airway soft tissues, while Whites showed evidence of both bony and soft tissue abnormalities ²⁴.

Three of the four relevant studies among children or adolescents regarding OSA and BMI found significant racial differences ^{77,98,109,112}. For instance, Johnson et al. found that more children with SDB were obese, and Black children who were obese were over 2 times more likely to have SDB, and the association between BMI and SDB was significantly greater for White than Black adolescents ⁷⁷. Another study found that upper and lower respiratory problems and obesity were risk factors for SDB in children and adolescents, and that increased risk of SDB in Blacks appears independent of the effects of obesity or respiratory problems ⁹⁸. Stepanski et al. concluded that Black children with SDB may be at increased risk for cardiovascular consequences due to greater oxygen desaturation levels with obstructive events compared to White and Latino children ¹⁰⁹. The authors also concluded that the role of obesity as a risk factor for OSA increases in children above 8 years old. A separate study found that the association between BMI and SDB – based on loud snoring, gasping/choking or snorting, awakening with gasping or choking, or momentary periods of stopped or abnormal breathing occurring weekly – was 2-fold higher among White adolescents compared to their Black counterparts ¹¹².

Insomnia and Obesity

In terms of insomnia and obesity among adults, Lee et al. observed that insomnia was significantly associated with greater BMI for Black, but not White Veterans ⁸⁹. The study concluded that insomnia (and posttraumatic stress disorder) may contribute to the development of health problems related to weight among Black veterans, and that early intervention may reduce this traditionally under-resourced population's risk. Subramanian et al. discussed the influence of gender and race/ethnicity on the prevalence of insomnia in patients with OSA, and explored the association between these two disorders with an emphasis on psychophysiologic insomnia ¹²³. Based on this review including White, Black, and Hispanic women, White women were more likely to have self-reported sleep maintenance insomnia, and Hispanic women were more likely to complain of insomnia related to psychophysiological symptoms.

Inconsistent findings could be due to different 1) intra- and inter-group study population characteristics; 2) study designs; and 3) sampling strategies including non-validated self-reported vs. objective sleep measures. From an epidemiological perspective, observed racial differences are either artifactual (due to random variability, differential bias across strata, differential confounding) or factual (due to actual differences in host factors) ¹²⁴. Some researchers consider innate racial differences – despite widespread acceptance of its nature as a social construct – without adequate conceptualization of and control for the nuanced artifactual possibilities (e.g. environmental and social factors that differ between groups). The following section describes potential reasons for racial/ethnic disparities in the sleep-obesity relationship.

POTENTIAL REASONS FOR RACIAL/ETHNIC DISPARITIES

All human sleep needs likely vary between individuals, but we know - from animal, clinical, and epidemiological studies - that social and environmental factors like light, temperature, nutrition and physical activity timing as well as quality, medication use, and even psychosocial stress can all influence sleep duration, quality, and timing. Inopportune exposure to these and other environmental factors can lead to disturbed sleep. For instance, a stressed individual is less likely to experience deep or N3 sleep (the physiologically restorative stage). Since racial groups have historically and continue to live in largely different physical and social environments, these long-term/intergenerational racial differences in inopportune exposures could lead to differential risk of physiological dysregulation due to suboptimal sleep. Physiological dysregulation resulting from poor sleep may subsequently contribute to the disproportionately increased risk of obesity-related conditions along with its cascade of health consequences. For example, many racial/ethnic minorities may be more likely to experience short sleep, which can lead to dysregulation of the aforementioned appetite-regulating hormones that increase food consumption and cravings for sweet and salty foods and/or activation of brain regions associated with overeating, thereby leading to an increased risk of obesity. In terms of other health conditions for which obesity is an established risk factor and sleep may contribute, hypertension risk may be higher among Blacks possibly due, in part, to 1) non-dipping of blood pressure during deep or N3 sleep; 2) type 2 diabetes risk may be greater due to greater levels of insulin resistance; and 3) cardiovascular disease due to, as an example, greater levels of inflammation. Moreover, stroke mortality is twice as high, and end-stage renal disease is 5 times higher among Blacks compared to Whites, and sleep could play an important, understudied role in differential risk of these health conditions.

CONCEPTUAL FRAMEWORK FOR DISPARITIES IN THE SLEEP-OBESITY RELATIONSHIP

Considering previously observed associations between sleep and obesity and how the relationship may differ by race, it is important to identify the fundamental or root causes of racial differences in the relationship to fully understand and intervene in order to address widespread health inequities. As illustrated by Figure 4, there are three main upstream factors that could contribute to racial disparities in the sleep-obesity relationship – the social and physical environment, genetics, or gene-by-environment interactions.

Environment

Regarding the physical and social environment, would we expect the sleep-obesity relationship to be the same between races if racial groups lived in the same environments? Blacks or African Americans, for example, are and historically have been disproportionately represented in lower socioeconomic status groups. Compared to Whites, Blacks have greater exposure to potentially health damaging environments and less access to health promoting goods and services that may influence risk of both suboptimal sleep and obesity or their common causes¹²⁵. In fact, sleep is not entirely physiological or endogenous and is responsive – either positively or negatively - to exogenous factors like environmental light,

noise, temperature, psychosocial stress, medication use, as well as quality and timing of both food consumption and physical activity, which are differentially experienced by race and socioeconomic status. Moreover, environmental changes have been implicated in the obesity epidemic as endogenous factors like genetics cannot explain the apparent decline in sleep and relatively rapid rise in obesity. Some studies have also shown that when racial groups live in the same [low-income] residential environments, chronic conditions like obesity and even behaviors are the same ^{126,127}.

Genetics

Genetics is another important upstream factor as genes are believed to account for 31 to 55% of a person's sleep duration with behavior and environment accounting for the remainder ¹²⁸. The billion-dollar human genome project revealed that all humans are 99.9% the same (genetic variants overlap) and that there is more genetic diversity within socially-constructed racial categories (especially among people of African ancestry) than there is between the racial categories. Despite the overlap of genetic variants across racial groups, future research should investigate how much of the observed differences in various sleep characteristics that affect obesity risk are explained by ancestral differences in genetic architecture (e.g. haplotypes, structural variation) while acknowledging that human behaviors like sleep are complicated and strongly influenced by non-genetic factors. Admixture mapping studies are capable of revealing potential ancestral differences in genetic variants associated with OSA ¹²⁹.

A meta-analysis suggests that the FTO fat mass and obesity-associated gene may have low-penetrance with limited evidence of a different prevalence by racial group ¹³⁰. Another study (that needs to be replicated) concluded that a polymorphism most frequent among those of African ancestry was associated with acquiring less N3 sleep, which is important for physiological restoration ¹³¹. In terms of biological differences, craniofacial features like nasal airways and tongue mass (along with other vulnerabilities making groups more susceptible to adverse events) may also affect overall differences in the restorative nature of sleep, and these craniofacial differences may determine a large portion of the predilection of OSA to Asian Americans ²⁴.

Nonetheless, many scientists agree that race – distinct from ancestry – is a social construct in that phenotypes like skin and eye color are not innately, strongly linked to health conditions with mainly lifestyle-related causes ¹³². Race could rather serve as a proxy or marker for relative advantage or disadvantage by indicating groups with more or less social and economic obstacles to health. While genetics and biology clearly have a strong influence on sleep in all individuals, they are unlikely to be the main reason for differential risk of suboptimal sleep and obesity between races, and modifiable environmental factors are likely to play a larger role than single nucleotide polymorphisms with minor allele frequencies (that generally need to be functional and may still confer small risks) in shaping overall and racial differences in variation in health and disease.

Epigenetics

Lastly, the field of epigenetics or gene-by-environment interactions has revealed that gene expression can be modified without altering an individual's actual genetic code, and environmental factors like nutrition and toxicants (e.g. phthalates, bisphenol-A, fungicide vinclozolin) promote the epigenetic transgenerational inheritance of disease and phenotypic variation¹³³⁻¹³⁵. Epigenetics may play an important role in racial differences in the sleep-obesity relationship since Blacks and Whites have been exposed to very different physical and social environments across lifespans and for many generations due to historical phenomena like slavery and other even more contemporary discriminatory policies/practices. Racial differences in early-life and lifetime stressors may be particularly important sources of epigenetic changes that can lead to differential obesity risk later in life. Mediators like biological mechanisms and behaviors along the pathway between sleep and obesity are likely affected by upstream environmental and social factors and could contribute to the often-observed racial differences in risk, prevalence, and severity of both sleep disorders like obstructive sleep apnea and insomnia.

Downstream, Intermediate, and Upstream Determinants

As a conceptual framework, I used the socioecological framework developed by the National Cancer Institute's Centers on Population Health and Health Disparities, which is described in greater detail elsewhere¹³⁶. In brief, the framework draws upon the realization that individual behaviors, like sleep, are influenced by complex and dynamic interrelations between an individual and his or her physical and social environments, which are affected by even more upstream or root causes of disparities like modifiable social conditions and even policies. Acknowledging that a person's biology constantly interacts with their environment across their lifespan, in the following section, I discuss examples of downstream, intermediate, and upstream determinants relevant to sleep and obesity and that likely contribute to racial/ethnic disparities in the sleep and obesity relationship due to, for instance, confounding from differential exposure by race and socioeconomic status.

Downstream determinants of sleep health and obesity

Starting with downstream or individual-level determinants of health where most research is being conducted, many observational and experimental studies investigate biological responses to behaviors that have an impact on sleep and risk of obesity, and health disparities researchers generally investigate how biological responses to those behaviors differ by demographic groups like race and ethnicity, for example. Furthermore, we investigate how individual health behaviors like sleep hygiene (e.g., one's bedtime routine and use of technology or blue-light emitting devices before bed) and obesity-related behaviors like nutrition and physical activity influence one's sleep duration, quality, and timing. Although poorly understood, it is well-known that these characteristics differ by demographics like race/ethnicity and SES. Further mechanistic research using -omics may deepen our understanding of the biological processes by which sleep and circadian rhythms influence obesity-related health outcomes¹³⁷. For example, O'Keefe et al. conducted food exchanges between African-Americans given a high-fiber, low-fat African-style diet and rural Africans given a high-fat low-fiber western-style diet, and found – in comparison to

their typical diets – notable changes in microbiota and metabolome known to influence colon cancer risk ¹³⁸. Gut microbes may alter the sleep-wake cycle, affect sleep- and wakefulness-regulating hormones, and shift circadian rhythms while sleep may impact human microbiome diversity and, therefore, health.

If we in science and public health with interests in studying and addressing disparities continue to focus the vast majority of our efforts and funding on this end of the spectrum to understand biological responses to individual risk behaviors, then we will continue to limit our ability to address preventable health disparities because, as illustrated by the aforementioned framework, the fundamental causes of disparities are going unaddressed or under-investigated. Ultimately, biological investigations are necessary, but insufficient for health disparities researchers to be maximally effective.

Intermediate determinants of sleep health and obesity

It is believed that intermediate determinants of health like neighborhoods represent the physical and social environments where upstream determinants like socioeconomic status or discrimination, for example, are realized or experienced by linking the physical and social environments to individuals and, thereby, influencing more downstream determinants like sleep-related risk behaviors as well as biological responses (e.g. weight gain/obesity) to those risk behaviors. For example, neighborhood and housing disadvantages like inopportune light and noise and neighborhood disorder could influence sleep health and contribute to health disparities ¹³⁹. Obesity could be affected by the neighborhood's walkability (or ability to engage in functional or leisure-time physical activity) and the food environment like food deserts being more prevalent in low-income, minority communities ^{140,141}. Just as the built environment (e.g. neighborhoods) can affect obesity risk, they can also influence how restorative sleep is for an individual through several pathways, including stress from disorder and the food environment through access to healthy food (acknowledging a bidirectional relationship between quality of nutrient intake and sleep health/disorders), places to exercise, and cultural differences. Housing conditions like the quality of the immediate sleep environment are also important, and likely differs by race through differential socioeconomic status and cultural influences.

Work environments as an intermediate determinant are also important for sleep and obesity disparities because long work hours, nightshift work, work-related stress or job strain and effort-reward imbalance, commute times, the food environment in workplaces, and impaired work-life integration can influence behaviors like sleep, physical activity, and nutrition. These factors can be differentially experienced by race. For instance, job strain and even widespread racial residential segregation can contribute to longer commute times and different transportation modes by race. Also, we found that the prevalence of short sleep duration by occupational class across various industries of employment generally increased with decreasing professional roles among Whites but the opposite was true for Blacks where Black professionals had the highest prevalence of short sleep of anyone, including their non-professional Black counterparts ⁹². In a separate study, we also found evidence of important, socially-patterned disparities in obesity by occupational class and across industries ¹⁴². As another example of an intermediate determinant, social relationships,

family routines and parenting styles could additionally influence sleep early in life because children usually take on the sleeping routines of family members ¹¹⁴. Structural factors like parental work schedules likely also influence children's sleep especially if the parent works multiple jobs including night shift and isn't home to, for example, enforce curfews or bedtimes. The literature supports an association between adverse childhood experiences (like physical and sexual abuse) with risk of sleep disorders in adulthood ¹⁴³, which may reflect several pathways related to disrupted neurodevelopment and failure to develop a healthy sleep schedule. Adverse childhood experiences are also associated with increased obesity risk ¹⁴⁴.

Upstream determinants of sleep health and obesity

Upstream determinants like poverty, socioeconomic status, and discrimination are considered fundamental or root causes of health disparities because their influence contributes to variation in health and disease. For example, discriminatory practices or policies that help funnel certain demographic groups into impoverished neighborhoods with high crime and stress (largely due to poverty) could contribute to sleep and obesity disparities especially compared to groups less affected by discriminatory practices or policies. Discrimination is an important upstream determinant because it shapes societal policies, practices, and norms in ways that can affect obesity across the lifespan through inequities in access to, for example, higher education, economic opportunity, healthy foods and health-promoting behaviors like sleep. Therefore, racial discrimination is considered a key determinant of racial disparities in health in general, and sleep is likely an important mechanism by which social factors get “under the skin” to influence disparities in obesity-related health. With regard to scientific evidence, a study found that Blacks who reported greater levels of racism-related vigilance (or bracing themselves in anticipation of experiencing racism) had greater levels of sleep difficulty compared to Whites ¹⁴⁵. Hispanics showed a smaller, but non-significant association, while Whites showed no association between racial vigilance and sleep difficulty. Unique stressors like racial discrimination among Black men and women may increase risk of obesity through disrupted sleep and increased food consumption. There is also data suggesting that perceived day-to-day interpersonal racial discrimination is associated with greater sleep disturbances and daytime fatigue as well as acquiring less deep or N3 sleep ^{146,147}. Another study found that multiple levels of racism, including interpersonal experiences of racial discrimination and the internalization of negative racial bias, operate jointly to accelerate vascular aging, which involves endothelial cells, smooth muscle cells and cardiomyocytes, as measured by telomere length ¹⁴⁸. Social factors like perceived interpersonal discrimination (a social pathway) may “get under the skin” to affect obesity risk by disrupting sleep duration and quality (behavioral pathways), which could lead to physiological or cellular dysregulation (a biological pathway) that eventually leads to compromised obesity-related health and well-being.

FUTURE RESEARCH DIRECTIONS

This section includes recommendations for 1) advancing research methodology, 2) future research topics, as well as 3) some of the policies and structures needed to investigate and eventually address racial/ethnic disparities in the sleep-obesity relationship (see Table 2).

Regarding research methodology, investigators should prioritize prospective or longitudinal studies to address the limitations of using the preponderance of cross-sectional data related to sleep and obesity. Therefore, existing and future studies need to include repeated objective measures of sleep and obesity that are ideally validated and standardized for the population of interest. There are various important research questions – relevant to disparities research – that the field has yet to answer and that require experimental or randomized controlled trial designs. For instance, we do not know if extending sleep in short sleepers translates into better [obesity-related] health outcomes. Observational studies alone would not be able to answer this important research question. Sleep, in its various dimensions (e.g., duration, quality, satisfaction), should be measured and incorporated into existing laboratory, clinical, epidemiological studies with population-based and prevention approaches, and weight management programs. Furthermore, mixed methods (quantitative and qualitative data) and community-based participatory research should be employed. Machine learning techniques may be particularly helpful for identifying novel contributors. The inclusion of racial/ethnic minority and female participants in well-powered studies where differences by racial groups and sex are explicitly investigated are also needed. Of note, two separate sex-specific mechanisms explaining the effects of sleep restriction on food intake have been proposed since data suggests a particular effect on appetite among men and another effecting satiety in women ¹⁴⁹.

With regard to suggested research topics for evidence-based public health recommendations, future studies should investigate concordance between objective and subjective sleep measures since most epidemiologic studies rely on self-reported data with minimal understanding of how it reflects objective measures (especially by race). Studies also need to investigate life-course and critical periods like early-life social and chemical exposures. Social and environmental determinants of sleep health and obesity, structural/non-structural factors that contribute to racial differences in time use (sleep opportunity), and daily stressors that affect sleep and obesity are also warranted. Considering that upstream environmental factors can positively or negatively influence downstream biological processes related to both sleep and obesity, future research and interventions should investigate and intervene upon the established and potential exogenous circadian or sleep disruptors that exist where people live, work, play, and worship. Since racial/ethnic minorities are generally more likely to live in suboptimal conditions, this approach could provide important information regarding dynamic interactions that contribute to preventable differences in the sleep-obesity relationship. It is also important to investigate timing of food consumption relative to sleep, the independent contribution of sleep duration variability to obesity risk, place vs race, the exposome (especially at the microbiome and metabolomics) and sleep, as well as identifying biomarkers for sleep. More ancestrally diverse populations need to be incorporated into genome-wide association studies to ensure that health

disparities are not widened by more predictive, potentially therapeutic information being available for people of European descent¹³².

There are also broad policies and structures needed to address health disparities. For instance, future studies should employ, from a life-course perspective, a socioecological and biopsychosocial vs. biomedical perspective. Unequitable distribution of power, resources, prestige and discriminatory policies and practices – as fundamental causes of health inequities – need to be studied in order to be addressed. A health and health equity focus in all policies using health impact assessments and health equity impact assessments could help address both sleep and obesity disparities. Interventions that focus on structural and individual-level changes that are culturally-relevant and place-based (given widespread racial residential segregation) should be considered, and the community should be involved from the outset. Since sleep literacy is low, efforts should be focused on increasing public awareness of the importance of sleep through, perhaps, implementation and dissemination science. In addition to increasing diversity in the workforce, sleep medicine education should be integrated into medical training and sleep specialists in federally qualified health centers.

CONCLUSION

Disordered sleep and obesity, disproportionately impacting many non-White populations, are socially patterned and embedded in physical as well as social contexts that are influenced by even more upstream modifiable social conditions. There is widespread acceptance that modifiable factors in the physical and social environments are important drivers of both poor sleep and obesity in a bidirectional manner; therefore, social determinants of health should be considered major potential contributors to disparities in both sleep and obesity. Also, early-life exposure or experiences seem particularly important since exposures during developmental periods may set individuals on a trajectory for compromised or poor health, and racial/ethnic groups should not be studied as a monolith (whenever possible) because important variation (e.g. socioeconomic) within racial groups exist. Ultimately, researchers should conduct future studies that address gaps in our current understanding as well as implement and evaluate place-based environmental interventions for sleep in hopes of improving population health overall while addressing health disparities.

ACKNOWLEDGEMENTS

I am grateful to Dr. Susan Redline for her thoughtful comments on a prior draft of this review, and to Ms. Mai-Han Trinh as well as Mr. Orlando Davy for their assistance with preparation of tables for this review. This work is funded by the Intramural Program at the NIH, National Institute of Environmental Health Sciences (Z1AES103325-01).

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Figure 1. Potential Biological Mechanisms Linking Sleep Restriction to Obesity

Adapted from Zimberg et al. Cell Biochem Funct 2012

Reference: DOI-[10.1002/cbf.2832](https://doi.org/10.1002/cbf.2832)

Accession number: 22473743

Figure 2. Obesity and Overweight Rates for Adults, National Health and Nutrition Examination Survey, 2013 to 2014²¹ (with Native American/Alaska Native Rates per 2014 National Health Interview Survey²²)

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Smoothed Lines of Mean Body Mass Index for Black and White Men by Educational Attainment, 1997-2008

Figure 3. Obesity Trends Among Black Men and Women

Figure 3 for men and women - merged as one for this review.

Reference - Jackson et al. Black-white disparities in overweight and obesity trends by educational attainment in the United States, 1997-2008. *Journal of Obesity*. 2013

DOI- [10.1155/2013/140743](https://doi.org/10.1155/2013/140743)

Accession number - 23691282

Figure 4.

Conceptual Framework for Racial/Ethnic Disparities in the Relationship between Sleep and Obesity

Table 1. Characteristics of studies that investigate the sleep-obesity relationship and include racial/ethnic minority participant adults.

Author (year)	Study design (experimental, observational, cross-sectional, or prospective)	Age	Sample size	Sex	Race/ethnicity	Sleep measure	Obesity measure	Disparities-related finding
Bidulescu (2010)	Observational, cross-sectional	30 to 65 y	1515	Men and women	Black	Self-reported PSQI and GSQ score computed as a sum of response values for the 7 components of the PSQI scale	Self-reported BMI	Worse global sleep quality was associated with a higher BMI. Sleep duration, disturbances, and daytime dysfunction had moderate associations with BMI. Among women, there was an increased likelihood of obesity for those participants with a worse sleep disturbance and with worse daytime dysfunction. Sleep quality was inversely associated with obesity among women but not among men.
Brook (2013)	Observational, cross-sectional	Mean: 32 y	815	Men and women	Black; Puerto Rican	Self-reported sleep duration	Self-reported BMI	Individuals with short sleep duration were more likely to be obese.
Chen (2016)	Observational, cross-sectional	45-84 y	2046	Men and women	White; Black/African American; Hispanic; Chinese	Measured SDB severity	Measured BMI and waist circumference	There were significant positive associations between (1) BMI and (2) waist circumference and AHI in the overall cohort as well as within each racial/ethnic group, which varied in magnitude. The increase in AHI corresponding with a 1-unit increase in BMI (and waist circumference) was highest for Chinese, followed by Whites, Blacks, and then Hispanics.
Cizza (2014)	Observational, cross-sectional	18-50 y	120	Men and women	White; Black; Other	Measured and self-reported sleep duration, sleep quality, sleepiness; measured sleep efficiency and respiratory disturbance index	Measured BMI, waist and neck circumferences, subcutaneous and visceral abdominal fat	Neck circumference (NC) was inversely related to measured sleep duration, but this correlation lost significance after adjustment for BMI. Participants with OSA had a larger NC even without BMI differences between groups.
Ford (2014)	Observational, cross-sectional	20 y (median 46 y)	13,742	Men and women	White; Black; Mexican American	Self-reported sleep duration for weekdays/work days	Measured BMI and waist circumference	Sleep duration was inversely associated with BMI and waist circumference in models that did not adjust for depression score and diagnosed sleep disorders and in models that excluded participants with depression or a sleep disorder. The association was particularly strong among adults aged 20-39 y. These associations held for many of the major demographic subgroups and did not differ by 3 major racial/ethnic groups or sex.
Fulop (2012)	Observational, prospective: 4 y	30-60 y	5301	Men and women	Black	Self-reported sleep duration, sleep quality, SDB	Measured BMI as well as waist and neck circumferences	Waist and neck circumferences (but not BMI) were associated with higher odds of sleep symptoms, sleep burden, and risk of OSA.
Grandner (2014)	Observational, cross-sectional	18 y (mean 49.3 y)	5649	Men and women	Non-Hispanic White; Black; Mexican American;	Self-reported sleep duration	Measured and self-reported BMI	Very short and short sleep was associated with objectively measured obesity. No elevated risk was found for long sleep duration. Racial/ethnic differences in patterns of risk varied by outcome

Author (year)	Study design (experimental, observational, cross-sectional, or prospective)	Age	Sample size	Sex	Race/ethnicity	Sleep measure	Obesity measure	Disparities-related finding
					other Hispanic/ Latino; Asian/ other			studied in that the relationship between very short sleep and obesity was strongest among Blacks.
Hairston (2010)	Observational, prospective, 5 y	18-81 y (mean 41.7 y)	1107	Men and women	Black; Hispanic	Self-reported sleep duration	Measured BMI and abdominal fat mass (visceral adipose tissue and subcutaneous adipose tissue)	Participants younger than 40 y of age with extremes of sleep duration were more likely to experience increases in BMI, SAT, and VAT fat areas over a 5-y period. Short sleep was associated with the greatest accumulation of fat in each depot. No association between sleep duration and change in fat measures was observed in those older than 40 y. The sleep duration association did not differ by racial group.
Jarosz (2014)	Observational, cross-sectional	18-40 y (mean 33.9 y)	69	Women	Black	Self-reported sleep quality, sleepiness, and fatigue	Self-reported BMI	BMI was positively associated with overall fatigue severity and the fatigue severity subscales. Although BMI did not show a significant relationship with global sleep quality, there was a significant positive association between BMI and the sleep latency.
Knutson (2011)	Observational, cross-sectional	36 to 44 y	5725	Men and women	Cuban American; Mexican American; Puerto Rican American	Self-reported sleep duration	Measured BMI, upper arm circumference, medial calf skinfold, triceps skinfold, iliac crest skinfold, and subscapular skinfold	Shorter sleep duration was associated with larger body size in Mexican Americans only. There was no apparent association between sleep duration and body size in either Cuban Americans or Puerto Ricans.
Knutson (2017)	Observational, cross-sectional	18-74 y	13,429	Men and women	Hispanic/Latino	Self-reported sleep/wake times, self-reported chronotype, sleep apnea monitor	Measured BMI	Among participants 35 y of age, a later wake time and sleep midpoint was associated with lower BMI. Subgroup differences in sleep timing but not sleep-BMI association. None of the interaction terms between the sleep timing variables and Hispanic/Latino background or sex were significant.
Lee (2017)	Observational, cross-sectional	Mean: 50 y	171	Men and women	Black; White	Self-reported insomnia symptoms	Measured BMI (from medical records)	There was a positive association between insomnia (as well as post-traumatic stress disorder) and BMI among Black but not White veterans.
Madan (2012)	Observational, cross-sectional	18 to 53 y (96%; 18 to 24 y)	402	Men and women	Native Hawaiian/Pacific Islander; Asian American; White	Self-reported sleep difficulties	Self-reported BMI	Sleeping difficulty was associated with greater odds of obesity.
Mokhlesi (2012)	Observational, cross-sectional	18 to 88 y	1019	Men and women	White; Black	Measured sleep architecture	Measured BMI	Overweight and obese individuals experienced less slow-wave sleep than their normal-weight counterparts, even after adjustment for OSA severity. Those with severe OSA experienced less slow-wave sleep than those with less severe OSA, even after

Author (year)	Study design (experimental, observational, cross-sectional, or prospective)	Age	Sample size	Sex	Race/ethnicity	Sleep measure	Obesity measure	Disparities-related finding
Montag (2017)	Observational, cross-sectional	35-64 y	492	Men and women	White; Black; Hispanic; East Asian	Measured and self-reported sleep duration; measured sleep fragmentation; self-reported sleep quality and sleepiness	Measured BMI	adjustment for obesity. There was no difference in OSA severity by race. There was no association between objective sleep duration, self-reported daytime sleepiness, or global sleep quality and odds of obesity. There were significantly greater odds for obesity associated with higher sleep fragmentation only in the unadjusted model. Race/ethnicity and sex did not significantly modify the relationships of interest.
Mosca (2012)	Observational, cross-sectional	40 y	371	Men and women	White; Hispanic; Black; Asian; other	Self-reported sleep duration	Measured BMI and waist circumference	Snoring was significantly associated with being overweight/obese. There was no significant interaction noted for race/ethnicity.
Owens (2010)	Observational, cross-sectional	45 to 78 y	224	Men and women	Black; White; Asian	Measured and self-reported sleep duration and sleep efficiency	Measured BMI and waist circumference	Napping in middle-aged men and women was associated with less nighttime sleeping in Blacks compared with Whites. Napping was associated with lower sleep efficiency as well as increased BMI and waist circumference. The addition of AHI as a covariate did not alter the results. There were no interactions with race/ethnicity for napping and cardiovascular disease risk factors.
Patel (2015)	Observational, cross-sectional	18-74 y	11,860	Men and women	Puerto Rican heritage; other Hispanic group	Measured sleep apnea; self-reported sleep habits, sleep duration, sleepiness	Measured BMI	Short sleep, highly variable by US Hispanic/Latino heritage, was significantly associated with increased odds of obesity.
Pedraza (2012)	Observational, prospective: 3 y	75 y	1085	Men and women	Mexican American	Self-reported quality of sleep	Measured BMI	Obesity predicted waking up several times per night.
Redline (2014)	Observational, cross-sectional	18-74 y (mean 45.9 y)	14,440	Men and women	Hispanic/Latino	Measured sleep apnea; self-reported sleep habits and sleepiness	Measured BMI and waist circumference	SDB was more common in overweight and obese individuals and increased progressively across ages 18-69 y.
Yu (2016)	Observational, cross-sectional	18 (mean 43.7 y)	535	Women	White; Black; Hispanic; other	Self-reported sleep disturbance	Measured BMI	Sleep disturbance mediates relationship depression and obesity.

AHI, apnea-hypopnea index; BMI/z, body mass index z-score; WHtR, waist-to-height ratio; SAT, subcutaneous adipose tissue; VAT, visceral adipose tissue; EEG, electroencephalogram; [PSQI](#), [Pittsburgh Sleep Quality Index](#); GSQ, Global Sleep Quality; BMI, Body Mass Index; SDB, Sleep Disordered Breathing; y, year; OSA, Obstructive Sleep Apnea.

Table 2.

Characteristics of studies that investigate the sleep-obesity relationship and include racial/ethnic minority participant children or adolescents.

Author (year)	Study design (experimental, observational, cross-sectional, or prospective)	Age	Sample size	Sex	Race(s)	Sleep measure	Obesity measure	Disparities-related finding
Boles (2017)	Observational, cross-sectional	Parents: 18 y; children: 3-5 y	75	Men and women; boys and girls	Hispanic; Black	Self-reported Child Sleep Habits Questionnaire; 7-Day Sleep Diary	Measured BMI	There were no significant relationships among the parent's BMI, child weight, and child sleep variables, including parent-reported sleep duration and sleep problems. Overall, the statistical model for Whites had a greater number of significant pathways compared with Blacks, but hours of sleep seemed to influence obesity in the model for Blacks but not in the model for Whites.
Dodor (2010)	Observational, cross-sectional	14 to 18 y	14,738	Boys and girls	White; Black	Self-reported sleep duration	Self-reported BMI	
Fleary (2017)	Observational, cross-sectional	16 y	14,815	Boys and girls	White; Black; Asian; other (Alaskan Native/American Indian, Native Hawaiian/Pacific Islander, multiple races); Hispanic, Hispanic-multiple	Self-reported average sleep duration on school nights	Self-reported BMI	Insufficient sleep was a common characteristic across race-sex groups and did not differentiate the groups in terms of obesogenic behaviors.
Ford (2016)	Observational, cross-sectional	3-17.9 y	7956	Boys and girls	Non-Hispanic White; Black; Hispanic; Asian/Pacific Islander; other/unknown	Self- or parent-reported sleep duration	Measured BMI	Adolescents with severe obesity shared mostly the same unhealthy behavior trends (including inadequate sleep). Sleep behavior also differed by race with a significantly greater proportion of Black and Hispanic children/adolescents reporting inadequate sleep hours per night.
Graef (2014)	Observational, cross-sectional	7.7 and 12.9 y	143	Boys and girls	White; Black; biracial/multiracial; Native Hawaiian; Asian	Measured sleep duration, sleep onset latency, and total wake minutes	Measured BMI and zBMIs	Among primarily obese children, Black (Hispanic and non-Hispanic) children had less sleep than Whites. Hispanic children had more sleep than non-Hispanics.
Haines (2013)	Experimental, baseline and 6-mo follow-up	Parents: 18 y; children 2-5 y	121 families	Men and women; boys and girls	Hispanic; Black; White/other	Parent-reported child sleep duration, bed time, wake time, and naps	Measured BMI	Promoting household routines, particularly increasing sleep duration and reducing TV viewing, was associated with a decrease in BMI.
Johnson (2006)	Observational, cross-sectional	13 to 16 y	1014	Boys and girls	White; Black	Parent-reported and self-reported SDB; self-reported sleep duration	Self-reported BMI	Black adolescents were over 2 times more likely than White adolescents to have SDB. The association between BMI and SDB was

Author (year)	Study design (experimental, observational, cross-sectional, or prospective)	Age	Sample size	Sex	Race(s)	Sleep measure	Obesity measure	Disparities-related finding
Kuo (2015)	Observational, prospective, 7 y	mean: 19.6 y	246	Late adolescent boys and girls	Mexican American	Self-reported sleep duration	Self-reported BMI	significantly greater for White than Black adolescents. There was a negative association between short sleep duration and BMI after accounting for prior BMI. Sleep variability was not associated with BMI.
Lumeng (2007)	Observational, cross-sectional	9 to 12 y	785	Boys and girls	White; non-White	Maternal report on the Children's Sleep Habits	Measured BMIz	Participants with shorter reported sleep duration in 6th grade were more likely to be overweight in 6th grade. Those with shorter sleep duration in the 3rd grade were also more likely to be overweight in the 6th grade, which was independent of the child's weight status in the 3rd grade. Sleep problems were not associated with overweight.
Martinez (2014)	Observational, longitudinal–2 y	8–10 y	229	Boys and girls	Mexican American	Measured sleep duration	Measured BMIz, waist-to-height ratio, and weight gain	Children who slept less were more likely to have a higher BMIz, WHtR, and weight gain at the 24-mo follow-up.
Redline (1999)	Observational, cross-sectional	2 to 18 y	399	Boys and girls	Black; White	Measured and self-reported sleep	Measured BMI and neck circumference	Moderate SDB was associated with increased odds of obesity. Increased risk in Blacks appears to be independent of the effects of obesity or respiratory problems.
Reither (2014)	Observational, cross-sectional	Mean 16.1 y	30,133	Boys and girls	White; Hispanic; Asian; Black	Self-reported sleep duration	Measured and self-reported BMI	Sleep duration was negatively associated with BMI among White, Hispanic, and Asian boys; positively associated with BMI among Black girls; and not related to BMI among Black boys or girls from White, Hispanic, or Asian ethnic groups.
Rudnick (2007)	Observational, cross-sectional	2 to 19 y	299	Boys and girls	Black; White; Asian; Hispanic	Self-reported SDB	Measured BMI/body fat percentage	Compared with patients from a general pediatric clinic, more children with SDB were obese or underweight. Among Black children, those who were obese were over 2 times more likely to have SDB.
Stepanski (1999)	Observational, cross-sectional	<17 y	198	Boys and girls	Black; White; Hispanic	Measured SDB, OSA	Measured BMI	Only the older children with SDB were heavier than age-matched normal sleepers. Children with SDB had increased EEG arousals, and sleep architecture was not otherwise significantly different from the non-SDB group. Black children with SDB had significantly greater oxygen desaturation with obstructive events compared with White and Hispanic children. Black children with SDB may be at increased risk for hypoxemia and cardiovascular consequences of SDB.
Storfer-Isser (2012)	Observational, prospective: 8 y	8–11 y at baseline	313	Boys and girls	Blacks; non-Blacks (few	Parent-reported, self-reported, and	Measured and parent-reported BMI	Sleep duration at ages 8–11 y predicted BMIz in early and late adolescence for boys but not

Author (year)	Study design (experimental, observational, cross-sectional, or prospective)	Age	Sample size	Sex	Race(s)	Sleep measure	Obesity measure	Disparities-related finding
Wong (2013)	Observational, cross-sectional	9-12 y	483	Boys and girls	Hispanics and Asians	measured sleep duration		girls. The associations were largely attenuated after BMIz at age 8-11 y adjustment.
					Hispanic; Black	Measured and self-reported sleep duration	Measured BMI	Hispanic children slept 0.2 h/d longer than Black children. Obese children slept 0.2 h/d less than normal-weight children.
Zhou (2015)	Observational, prospective (2 y)	3-24 mo	899	Boys and girls	Chinese; Malay; Indian	Mother-reported infant sleep duration	Measured BMI and body length	Overall, sleep duration was significantly associated with body length but not BMI. Only in Malay children was shorter sleep associated with a higher BMI and shorter body length. In addition, shorter sleep was associated with a higher BMI and shorter body length in children who slept 12 h/d at 3 mo of age.