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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 nonmodifiable reasons. Because many people of color have historically lived and continue to largely live in disadvantaged, obesogenic physical and social environments, these greater adverse exposures likely negatively affect sleep, resulting in physiological dysregulation. Physiological dysregulation may, in turn, lead to increased obesity risk and subsequent health consequences, which are 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 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.

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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 insomnia.[1] Although a matter of debate, sleep duration appears to be decreasing, with Americans reporting, on average, 6.1 hours of sleep per day,[2–4] and lack of sleep has been shown to cost the US economy approximately $411 billion per year.[5] Sixty-million prescriptions for sleeping pills are written per year, and more than 300,000 (6400 fatal) accidents occur annually, which are largely attributed to drowsy driving.[6] Daytime sleepiness and difficulty falling asleep affect nearly 35% to 40% of US adults per year [7] 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 mortality.[8] The Institute of Medicine has identified sleep as an unmet public health need, and the Department of Health and Human Services has established 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 individuals.[1] Currently, 35% of American adults report habitual short sleep and 8% report being long sleepers.[9]

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, are 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 occur, growth and other metabolically active hormones are released, blood pressure normally drops, and breathing slows down. REM sleep has been associated with cognitive restoration, which is important for learning, memory consolidation, and emotion regulation.[13] Sleep duration

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recommendations differ across the lifecycle, and because 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 for sleep to be fully restorative.[14] A more recent consensus statement from the American Academy of Sleep Medicine and Sleep Research Society has 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 recommendations.[15]

Public health importance of obesity

Second to cigarette smoking, obesity is considered the second leading cause of preventable death and annually costs the US economy approximately $147 billion.[16] Thirty-five percent of US men and 40% of women are classified as obese,[17] 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 diabetes,[20] cardiovascular disease,[21,22] and premature mortality.[23] 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 (eg, cortisol) and exogenous (eg, nutrition, physical activity, psychosocial stress) factors independently affect both sleep and obesity,[25] and many organ systems (eg, endocrine, nervous, reproductive) are affected by both sleep and obesity. In fact, the prevalence of both suboptimal sleep and obesity has increased over the past several decades,[26] and they have common risk factors (eg, stress) as well as physical and mental health (eg, depression) consequences. Suboptimal sleep characteristics and sleep disorders (independent of body mass index [BMI]) are associated with hypertension,[27] diabetes,[25] cardiovascular disease,[28] and mortality.[29,30]

Regarding age, associations between sleep and obesity have been consistently stronger in children compared with adults.[23] Children and adolescents may be more vulnerable to the effects of insufficient sleep because 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 digestion.[31] A circadian rhythm for RMR exists which also varies by stage of sleep.[32] 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 obesity.[33]

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 hormones.[34] 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 adiposity.[35] 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 (Fig. 1).[10,36] Not getting enough sleep is associated with the downregulation 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 concert 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 food.[41] 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 an energy surplus. Multimedia use like television watching can contribute to sleep deprivation, increased sedentary behavior, and increased energy intake through, for example, exposure to advertisements.[42,43] Stress is also generally associated with consuming more energy overall as well as consuming a less healthy dietary pattern.[44] Compared with their well-rested counterparts, people with insufficient sleep have been shown to consume approximately 300 more calories (or 1255 joules) a day, with most of the additional energy coming from carbohydrates or high-fat foods.[45] Average weight gain is 0.5 to 1 kg per year,[46] 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 and cholesterol, and delayed or nighttime consumption is associated with decreased fat oxidation and increased cholesterol.[47]

With regard to social pathways, we all relatively recently started living in a 24-hour society that—with the advent of artificial electric light and advancing technology—can contribute to less time spent sleeping.[48] 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 to increase obesity risk by 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 may also make the affected individuals more vulnerable or susceptible to insufficient sleep duration and inadequate sleep quality.[49–53]

Racial/ethnic disparities in sleep and common sleep disorders

This section summarizes racial/ethnic disparities in sleep duration, quality, and the 2 most common sleep disorders, obstructive sleep apnea (OSA) and insomnia. Compared with 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

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