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The Epidemiology of Sleep and Obesity

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Abstract

Sleep is a state of consciousness that is preserved across animal species whose exact function is not yet clear but which has a vital impact on health and well-being. Epidemiological evidence suggests sleep duration in both children and adults has been decreasing over the past half-century, while at the same time, rates of overweight and obesity have been increasing. Short sleep duration along with other dimensions of poor sleep have been associated with obesity both cross-sectionally and longitudinally. These data suggest a potential causal relationship between poor sleep and greater rates of weight gain that may be related to effects of sleep on dietary intake or physical activity. However, there is also potential for reverse causation as obesity leads to many co-morbidities including sleep apnea that can disrupt sleep. Medium and long term interventional studies are needed to evaluate the potential for healthy sleep interventions to help combat the epidemic of obesity.

The prevalence of overweight and obesity has reached epidemic proportions in Western nations and increasingly, in developing nations as well. The growing rates of obesity have important health consequences, including increasing the risk of a host of diseases including degenerative joint disease, type 2 diabetes, cardiovascular disease, and obesity-associated malignancies. Public health efforts to combat obesity have focused on interventions to improve diet and increase physical activity. Recent data suggest that poor sleep habits may contribute to the risk of obesity, opening a new avenue for potential intervention. In this paper, we review the epidemiology of sleep habits in both children and adults including trends over time as well as evidence for an association between poor sleep habits and obesity.

Role of sleep

Sleep is a rapidly reversible recurring state of inactivity associated with diminished responsiveness to the external environment. It occurs in all multicellular animals, indicating its universal functionality.[1] Since an organism is unable to respond to external threats while

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asleep, there would be natural selection pressure to eliminate sleep if there was not a benefit to be gained from sleeping. Additional evidence for the biological importance of sleep is that there exists a homeostatic drive, which regulates sleep. Just as with hunger or thirst, the longer an organism goes without sleep, the sleepier it gets and the more likely the organism is to fall asleep. Further evidence of the vital role played by sleep comes from animal studies where complete sleep deprivation leads to death over a few weeks.[2]

Despite a clear understanding that sleep is vital to health, the exact role played by sleep remains controversial.[3] Among the leading theories for the role of sleep are that it provides a time of relative cortical quiescence to allow brain energy stores to rebuild,[4] that sleep allows an opportunity for synaptic pruning which facilitates neural plasticity allowing memory and learning,[5] and that sleep provides an opportunity for the clearance of neural waste products through the glymphatic system which is activated during sleep.[6] In addition to brain health, there is growing interest in the role sleep may play in somatic health. Growth hormone secretion during sleep may provide one mechanism for restoration of the periphery and regulation of immune system function may represent another.

Epidemiology of sleep duration

One of the simplest aspects of sleep to measure is duration, the total amount of time spent asleep. An increasing body of data suggests chronically insufficient duration of sleep is associated with poor health. Based on this evidence, in 2010, the US government for the first time set public health benchmarks to try to improve sleep duration as part of the Healthy People 2020 initiative.[7] In particular, goals have been set to increase the proportion of students in grades 9 through 12 getting at least 8 hours of sleep on school nights, the proportion of adults 18–21 getting at least 8 hours of sleep and adults 22 and over getting at least 7 hours of sleep per 24-hour period. For children and adolescents, the American Academy of Sleep Medicine currently recommends sleeping eight to sixteen hours per 24-hour period, with age-specific recommendations decreasing as age increases.[8] However, epidemiological data, which measures sleep duration via parent- or self-report, has found that younger and older children and adolescents in both the United States and other countries are not getting enough sleep.[9] Data from multiple years of the Youth Risk Behavior Surveillance System suggest that 95% of high school students are not meeting published sleep recommendations.[10] Across childhood age groups, sleep duration has declined by more than an hour over the past one hundred years.[11]

For adults, multiple expert panels have recommended at least seven hours of sleep per night,[12–14] but as of 2014, data from the Behavioral Risk Factor Surveillance System demonstrate that 33.8% of Americans were not meeting these recommendations.[15] In the United States, data from the National Health Interview Survey (NHIS) has shown that self-reported mean sleep time has decreased 10–15 minutes from 1985 to 2012, while the percentage of adults reporting less than 6 hours of sleep per night increased from 22.3% to 29.2%.[16] Possible explanations for the decreases in sleep duration include environment lighting, new technologies, and for adults, long working hours.[17]

Correlates of short sleep duration

Demographic factors

Short sleep correlates with several demographic characteristics. Sleep duration declines with age by about ten minutes per decade.[18–20] Sex differences also exist, as men have shorter sleep duration than women by approximately 25–45 minutes,[18,19,21–23] possibly due to hormones or sociological factors such as gender roles.[24] Variation by race/ethnicity is present, with African Americans generally sleeping shorter amounts than other racial/ethnic groups.[21–23,25–30] Short sleep has also been associated with lower education and income,[19,20,29,31] though some studies found no association.[21,27] Compared to those who are married, unmarried individuals are more likely to have insufficient sleep.[20,27,32]

Other factors

Other potentially modifiable factors have been associated with short sleep duration in cross-sectional analyses of nationally representative and community-based studies.

Neighborhood factors, such as the social and physical environment, have been examined with objectively and subjectively measured sleep duration.[33–35] In the Multi-Ethnic Study of Atherosclerosis, aspects of the social environment, such as disorder, social cohesion, and safety were associated with longer objective and subjective continuously measured sleep duration,[33,36] while in the Hispanic Community Health Study/Study of Latinos, the prevalence of short sleep was higher in unsafe versus safe neighborhoods.[35] Compared to rural residents, those living in urban areas are more likely to report short sleep,[23] potentially due to noise and light pollution.

Aspects of *employment,* including status and work hours*,* have also been associated with sleep. Short sleep duration is more common among those working full-time, while those who are retired, not retired/not working, and working part time were more likely to be long sleepers.[20,37] Additionally, compared to working shorter hours, long work hours (50+ per week) have consistently been associated with shorter sleep duration.[38–40] Working multiple jobs has also been associated with shorter sleep duration compared to working one job[41] and those with longer commutes compared to shorter are more likely to report short sleep duration.[40,41] Shift work, such as working overnight and early in the morning, has also been associated with insufficient sleep.[42]

In reviews of studies on children and adolescents, less total sleep time was associated with more television, computer, video game, and mobile device *screen time*.[43,44] In adults, more smartphone use was associated with short sleep duration,[45] though a Canadian study found co-occurring small increases in both average sleep duration and screen time from 1998 to 2010.[46] Exposure to blue light from screens in the hours before bedtime may suppress melatonin and result in circadian misalignment,[47,48] though short sleep could also lead to more screen time during waking hours.

Acculturation may be associated with objectively and subjectively measured short sleep in U.S. immigrant populations.[27,37,49,50] Compared to Mexico-born U.S. immigrants, Mexican Americans born in the United States are more likely to be short sleepers.[27,49] Additionally,

Hispanics and Latinos who had lived in the United States for a longer period of time were more likely to have shorter actigraphy-measured sleep duration,[37] while those in primarily English-speaking households were more likely to report short sleep than those in exclusively Spanish-speaking households.[27] These findings are suggestive of a "healthy migrant" effect, where immigrants adopt poor health behaviors as they adapt to U.S. culture.[51]

Short sleep duration and obesity

As sleep has decreased over time, the prevalence of obesity has increased. As of 2011–2014, the prevalence of obesity was 37.7% for adults[52] and 17.0% for children and adolescents.[53] The combination of these co-occurring phenomenon with pathophysiologic evidence[54] have suggested that sleep and obesity may be related. There is now an extensive literature on the association between sleep duration and obesity, including several systematic reviews and meta-analyses.[55–59] Early work consisted of predominantly cross-sectional studies, providing no information about temporality. Additionally, many relied on self-reported measures of sleep duration, which are only moderately correlated with objective measures such as actigraphy,[60–62] with mean subjective sleep being almost an hour more. The definition of short sleep lacks standardization so categorizations are variable, making comparisons across studies difficult.

Despite differences in measurement and study design, research in children and adolescents has consistently found that short sleep duration is positively associated with obesity.[59,63] The short sleep-obesity relationship in adults has been less consistent, with positive, U-shaped, and no associations,[59] indicating the association may differ by age. This inconsistent relationship has been found in a variety of study designs. In the Nurses Health Study, the risk of incident obesity was 1.15 (95% CI: 1.04, 1.26) for those reporting less than five hours of sleep and 1.06 (95% CI: 1.01, 1.11) for those reporting six hours of sleep, compared to those who reported seven hours of sleep per night.[64] In the CARDIA and SWAN cohorts, two longitudinal studies that used actigraphy, no associations were found between short sleep duration and weight gain or incident obesity.[65–67] Additionally, a review of the few randomized controlled trials that manipulated short-term sleep duration found inconsistent measurement and high heterogeneity for several adiposity and energy balance outcomes, with the most suggestive findings for body weight.[58] More work is needed to establish whether the short sleep-obesity association is causal.

Long sleep has also been examined in relation to obesity, though few plausible biological pathways have been hypothesized.[68] Most studies reporting positive associations rely on self-reported measures of sleep, indicating that long sleepers may be reporting more time in bed but not more time spent asleep.[68] Confounding by depression and low socioeconomic status may also be an explanation for these findings.[69]

Short sleep may influence obesity through several potential pathways.[54] One mechanism is that short sleep duration leads to increased food intake.[70] Experimental studies have demonstrated that short sleep may lead to self-reported and biological changes in hunger and appetite, though early findings on leptin and ghrelin have not been replicated.[71–73] Hedonic eating rather than hunger-driven eating may be a more likely mechanism to explain the

association between insufficient sleep and increased food intake.[73] Brain imaging data suggest that sleep restriction may increase central neuronal responses to unhealthy foods, potentially leading overeating.[74] Short sleepers may also have more opportunities to eat because they are awake for more hours in the day. Insufficient sleep may also lead to fatigue, resulting in decreased physical activity and increased sedentary activity, which could lead to obesity. However, research on short sleep duration and energy expenditure and metabolism suggests no large effects, though there are a limited number of experimental studies.[75]

Other sleep domains and obesity

Sleep health has increasingly been recognized to encompass multiple dimensions of sleep, each when altered has been associated with cardiometabolic disease.[76] Besides sleep duration, sleep quality, efficiency, daytime sleepiness, timing, and variability, have been individually examined with obesity, albeit less frequently. These domains are sometimes weakly correlated, and can be associated with obesity independent of sleep duration.

Limited research suggests that greater *sleep variability,* usually defined as the standard deviation of actigraphy-assessed sleep duration*,* is associated with obesity in both children and adults.[77–81] Those with high sleep variability may experience a lack of synchronization in eating patterns due to variation in sleep. Alternatively, high sleep variability may be indicative of insufficient sleep certain nights of the week and compensation on other nights.[77] In cross-sectional studies, high sleep variability has been associated with higher daily energy intake[82] and sugar-sweetened beverage consumption[83] and children who have consistent sleep throughout the week have higher levels of physical activity.[84]

Late *sleep timing,* usually defined as the sleep midpoint, has also been associated with obesity in some studies,[85–87] but not all findings were robust.[77,88] Like sleep variability, sleep timing may influence obesity by shifting the timing of eating patterns. Compared to those with earlier sleep times, those with later sleep times are more likely to engage in breakfast skipping and after dinner snacking.[85]

Daytime napping as measured via actigraphy has also been positively associated with obesity in one study, with interaction by nighttime sleep duration.[77] Among very short sleepers (<5 hours), there was no association between daytime napping and obesity, while there was a small positive association for those with longer nighttime sleep duration. Like variability and timing, daytime napping may reflect irregularity in the synchronization of the circadian rhythm or reflect an irregular sleep schedule.

Low *sleep efficiency,* defined as the percentage of the sleep interval spent asleep*,* has also been associated with obesity.[78,89] Those with low sleep efficiency may experience selective deprivation of stage N3 sleep. This has been linked to abdominal obesity,[90] through such potential mechanisms as the autonomic nervous system and hypothalamic pituitary adrenal axis.[91]

Sleep quality, often measured with tools such as the Pittsburgh Sleep Quality Index (PSQI),[92] has been associated obesity in some studies,[93,94] but not others.[95] The PSQI includes multiple components similar to the previous described domains, including sleep

duration, habitual sleep efficiency, and daytime dysfunction, and thus may influence obesity through similar pathways.

Obesity comorbidities associated with sleep

Sleep disordered breathing

Although short sleep is hypothesized to influence obesity, obesity, usually defined as a BMI ≥ 30 kg/m^2, may affect aspects of sleep. *Obstructive sleep apnea* (OSA), the most common form of sleep-disordered breathing, is characterized by the repetitive partial or total collapse of the upper airway, and is a highly prevalent consequence of obesity. Strong dose-response relationships have been found between obesity and OSA, where a higher body mass index is associated with greater OSA severity.[96–104] In longitudinal studies, both high baseline weight and weight change have been associated with the speedier development of more severe OSA.[105–107] Randomized trials have also demonstrated that weight loss is associated with decreases in the severity of OSA.[108] Such an accumulation of evidence is suggestive of obesity's causal effect on OSA. While OSA had previously been thought to worsen obesity, recent data from randomized trials demonstrate that OSA treatment is associated with weight gain rather than weight loss.[109]

Excess body weight may exacerbate OSA through different pathways. Animal models suggest that parapharyngeal neck fat may directly compress the upper airway.[110] Human physiologic studies demonstrate a strong correlation between BMI and the passive collapsing pressure of the upper airway.[111] By displacing the diaphragm upwards, abdominal adiposity reduces lung volume and therefore downwards tracheal traction. This loss of "tracheal tug" has been associated with greater upper airway collapsibility.[112] Recent imaging studies also suggest obesity is associated with intramuscular deposition of fat in areas such as the tongue, which may not only narrow the upper airway, but also interfere with the upper airway dilating function of these muscles.[113]

Other obesity comorbidities

Besides OSA, other obesity comorbidities may also impact sleep. *Depression* has been consistently associated with both sleep and obesity, though the directionality is unclear. In longitudinal studies, depression and obesity have been associated in both adults and adolescents.[114,115] Obesity may influence depression through insulin resistance or diabetes, which may influence the brain.[116] Psychologically, obesity may lead to lower self-esteem, higher body dissatisfaction, and disordered eating,[117] which have been associated with increased risk of depression. Obesity and depression may also affect each other through inflammation,[118,119] and/or dysregulation of the hypothalamic-pituitary-adrenal axis.[120] Depression may also influence obesity through poor lifestyle behaviors, such as poor diet quality, high sedentary activity, and low physical activity.

Depression has also been linked to sleep disturbances. Both short and long sleep duration have been linked to depression in a meta-analysis of prospective studies.[121] Insufficient sleep has also been associated with poor treatment outcomes for depression.[122] Short sleep may lead to daytime sleepiness and/or fatigue, which has been associated with

depression.[123,124] Depression has also been linked to other sleep disturbances, particularly insomnia, though the relationship is likely bidirectional.[125]

Obesity has also been associated with gastroesophageal reflux disease (*GERD*).[126,127] In most studies, obesity has been positively associated with GERD symptoms, erosive esophagitis, and esophageal adenocarcinoma,[128] though some relied on self-report measures of height and weight. Increases in weight over time compared to no change have also been associated with a higher risk of GERD symptoms.[126] In obese individuals, adipose tissue may compress the stomach, leading to increases in intragastric pressure, disruption of the gastroesophageal junction, and relaxation of the lower esophageal sphincter, which may facilitate hiatal hernia formation.[128] Few studies have examined the potentially bidirectional association between short sleep duration and GERD,[129,130] especially in community or population-based samples.[131] Results have been mixed, as two studies found an association between short sleep and GERD while another found no association. GERD is typically worse when supine and can be a cause of sleep disruption.

Obesity is also an established risk factor for degenerative joint disease or *osteoarthritis*.[132–134] Compared to those of normal weight, obesity has been associated with risk of incident osteoarthritis[135] and the worsening of prevalent osteoarthritis,[136] while weight loss has been associated with lower risk[137] and reductions in pain symptoms.[138] Excess weight adds additional pressure to the joints, while weight loss can reduce joint loads. Arthritis pain is commonly associated with disturbed sleep and poor sleep quality.[139]

Obesity and overweight have been associated with *asthma* in both children and adults. [140] Both overall and abdominal obesity have been associated with risk of incident asthma,[141] while weight loss has been associated with improvement of asthma symptoms.[142] Those with obesity and asthma may represent a distinct phenotype that is more severe and harder to control.[143,144] Compared to normal weight asthmatics, obese patients with asthma respond less well to established asthma treatments.[145] Asthma symptoms tend to worsen at night and have been associated with impairments in sleep.[146,147]

Besides comorbidities, obesity may have a direct effect on sleep. In clinical samples, obese patients without sleep apnea are more likely to experience daytime sleepiness and nighttime sleep disturbances compared to those of normal weight.[148,149] Further, in the general population, obesity has been identified as a risk factor for excessive daytime sleepiness independent of both sleep apnea and depression, supporting the notion of a direct effect on obesity on sleep mechanisms.[98] In uncontrolled studies, bariatric surgery is associated with improvements in sleep quality and daytime sleepiness.[150] It is hypothesized that daytime sleepiness in obesity may represent effects of elevated pro-inflammatory cytokine levels or hypothalamo-pituitary-adrenal axis dysfunction.[151]

Conclusion

Short sleep duration and other aspects of poor sleep habits are growing in prevalence in modern society in both children and adults. A growing literature has established that short sleep duration and other dimensions of poor sleep are associated with obesity and appear to

predict obesity risk and rate of weight gain longitudinally. Small experimental studies suggest poor sleep may impact dietary intake particularly hedonic eating. However, because of the potential for confounding and reverse causation, it is yet unclear whether poor sleep actually causes obesity. Interventional studies that target improvements in sleep habits are sorely needed to assess the potential of sleep-focused therapies as tools in combating the obesity epidemic. Public health interventions such as delaying school start times provide opportunities to improve sleep health and may be used to test whether improving sleep has effects on obesity risk.

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