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Sleep and obesity risk in adults: possible mechanisms; contextual factors; and implications for research, intervention, and policy

Michael A. Grandner, PhD, MTR *

Sleep and Health Research Program, Department of Psychiatry, University of Arizona, 1501 N Campbell Ave, PO Box 245002, Tucson, AZ 85724-5002

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ABSTRACT

Obesity is a major public health problem among US adults. Insufficient sleep and sleep disorders are prevalent and may contribute to the public health problem of obesity. This review addresses several key questions regarding sleep and obesity in adults, including the following: (1) What constitutes adequate sleep in adults? (2) What are the consequences of inadequate sleep in adults? (3) What factors influence sleep in adults? (4) How can adults improve their sleep? (5) How can we implement these in adults? (6) How can these issues be addressed in future research and policy decisions? Although a comprehensive review of all of these is beyond the scope of this article, this review brings these concepts together toward a discussion of the role of sleep in the health of US adults.

Introduction

Obesity is a major public health problem[1] and a major risk factor for many of the leading causes of death.[2] This has prompted intense motivation to reduce the impact of obesity in our society. Yet, obesity rates continue to increase. Insufficient sleep has also been identified as a major public health problem,[3] as has the overlapping problem of sleep disorders. Many studies have examined relationships between sleep and obesity risk in adults.[4] This review will introduce several concepts that are relevant to this relationship, including sleep guidelines; contextual factors; and implications for intervention, policy, and future research.

What constitutes adequate sleep in adults?

Sleep is a biological requirement for human life. Yet, a clear understanding of how much sleep an individual needs remains elusive. It is likely that there is a distribution of sleep need in the population such that some individuals likely need more sleep than others. There is evidence of this from some genetics work,[5] but the few rare genes identified that are associated with shortened sleep in humans likely do not account for all of this variability.

Still, there is a lack of conceptual clarity regarding sleep "need"—need for what? It is not known whether a certain amount of sleep (of adequate quality, regularity, and timing) depends only on the individual or whether it depends on the outcome. Perhaps an individual requires a certain amount of sleep for optimal learning/memory but a different amount of sleep for optimal metabolism. The nature of these complex relationships is not known. Furthermore, there is evidence that independent of sleep "need" is a dimension of resilience to sleep loss,[6] so some individuals may be able to better cope with sleep that does not meet an individual's needs.

Guidelines and recommendations

Several organizations have published guidelines for healthy sleep in adults. The first published guidelines were from the National Sleep Foundation.[7] These guidelines, which were later followed up by a more detailed report,[8] used a structured process to assemble a consensus panel with representatives from a number of stakeholder organizations, review the literature, and come to conclusions regarding sleep recommendations for various age groups. The recommendation of this group was that adults should get 7-9 hours of sleep per night.

In parallel, the American Academy of Sleep Medicine and Sleep Research Society jointly convened a similar panel of scientists and clinicians who were content matter experts in sleep, as part of the National Healthy Sleep Awareness Project (http://www.sleepeducation.org/healthysleep). Using a similar structured process, their recommendations were published[9,10] and followed up by a final report.[11,12] Similar to the National Sleep Foundation guidelines, this group also

* Tel.: +1 520 626 6346.
E-mail addresses: grandner@email.arizona.edu, grandner@upenn.edu.

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recommended at least 7 hours for adults. However, this group did not reach consensus on an upper limit for recommendations.

Subsequent to these guideline documents, the American Thoracic Society and American Heart Association individually convened expert panels to develop position statements on sleep duration and quality. The recommendation of the American Thoracic Society was, similar to the National Sleep Foundation, that adults should get 7-9 hours of sleep.[13] The recommendation of the American Heart Association was, similar to the American Academy of Sleep Medicine and Sleep Research Society guidelines, that adults should get at least 7 hours of sleep,[14] with the addition that long sleep duration may be harmful but that the specific upper limit is unclear.

Since the publication of these guidelines, the Centers for Disease Control and Prevention have adopted "less than 7 hours of sleep" as a cut-off for "insufficient sleep."[15] This has been classified as an unhealthy behavior, similar to smoking, physical inactivity, and unhealthy diet.

What are the consequences of inadequate sleep in adults?

Many studies have used laboratory and survey methods to understand the consequences of inadequate sleep. Without a clear understanding of what an individual's "needs" are, 2 types of scientific approaches have attempted to address this question. First, laboratory studies have used sleep restriction/deprivation paradigms as a probe to observe consequences of acute sleep loss. Second, population studies have used epidemiologic surveys to explore associations between sleep reports and observed outcomes of interest. Both approaches have strengths and weaknesses (which have been discussed elsewhere[16,17]).

Over 50 years of population studies has documented that habitual sleep duration is associated with mortality risk.[18] These studies, which include millions of participants from multiple continents and many different study designs, systematically show that both "short" and "long" sleep (variously defined) are associated with increased mortality relative to those in the normative range.[19–21] Mechanisms for both short and long sleep have been proposed,[22] although findings are more consistent for short than long sleep. Obesity plays a prominent role in many of these proposed mechanisms as both a cause and a consequence of poor sleep.

Relationship with obesity

Nearly 100 studies have identified cross-sectional and longitudinal relationships between insufficient sleep and obesity (for reviews, see[4,23–32]). In a meta-analysis of prospective studies, Wu and colleagues[33] found that habitual short sleep duration was associated with increased risk of developing obesity (odds ratio [OR] = 1.45, 95% confidence interval [CI] 1.25-1.67). Some modifying factors may be relevant in this relationship. For example, studies using nationally representative data from the National Health and Nutrition Examination Survey found that the relationship between sleep duration depends on race/ethnicity (strongest results seen in blacks/African Americans[34]) and age.[35] Regarding age findings, among younger adults, more sleep was associated with lower body mass index; among middle-aged adults, both shorter and longer sleep durations were associated with higher body mass index; and among older adults, relationships were not seen. Thus, shorter sleep duration likely predicts incident obesity, although this relationship may be stronger among certain groups such as younger adults and Blacks/African Americans.

Cardiometabolic risk factors

Obesity often exists in the context of other cardiometabolic disorders. Many of these conditions have overlapping etiologies, and these various pathways may all contribute to obesity risk. Habitual short sleep duration has been associated with hypertension in a number of studies. Meta-analyses examining relationships between sleep duration and incident hypertension have shown effects in the range of OR = 1.17[23] to OR = 1.20.[36] Some studies have shown that short sleep duration is also associated with high cholesterol,[34,37,38] but studies showing a relationship with incident hyperlipidemia are lacking.

Insufficient sleep has also been linked with elevated systemic inflammation. Laboratory studies of sleep restriction have shown that sleep loss can acutely increase levels of C-reactive protein,[39] interleukin-6,[40] interleukin-17,[39] and tumor necrosis factor-α.[41] Population studies have also shown that short sleep can be associated with elevated levels of C-reactive protein,[42] interleukin-6,[43] tumor necrosis factor-α,[44] and other markers.[45] At the population level, relationships between sleep duration and C-reactive protein were shown to be independent of likely sleep disorders such as insomnia and sleep apnea.[42] Notably, many population studies find relationships between inflammatory markers and long sleep duration, sometimes in the context of no findings for short sleep.[45] In addition to its role as a marker of cardiovascular risk, inflammatory markers often play important roles in metabolic regulation, such as regulation of insulin secretion, and this could represent a pathway linking sleep loss and metabolic dysregulation, which could lead to obesity and/or diabetes.

At the population level, habitual short sleep duration is associated with incident diabetes. Results from recent meta-analyses estimate that habitual short sleep is associated with approximately 30% increased risk vs 7-8 hours of sleep.[46,47] Laboratory studies have attempted to understand mechanisms of these relationships. Several studies have found that acute sleep restriction can lead to physiologic conditions indicative of diabetes risk, such as insulin resistance[25,48] and impaired glucose tolerance.[49,50] Several studies have also shown that acute sleep restriction can impact physiologic signaling pathways that play a role in appetite regulation, including decreasing leptin,[25,51,52] increasing ghrelin,[25,27,51,53] and increasing endocannabinoids.[54] Furthermore, research into signaling molecules secreted by adipose tissue from both population and laboratory studies implicates sleep-dependent changes in adipose tissue as well.[55]

In addition to studies of acute sleep loss, another emerging literature explores the role of circadian genetics in expression of proteins that can influence metabolic processes. A recent review of this work can be found here.[56] In brief, circadian rhythm disruptions may cause disruptions in insulin signaling through disruptions of rhythmic transcription of gene networks in exocytosis driven by glucose, cyclic adenosine monophosphate, and calcium. In addition, melatonin, glucagon-like peptide 1, and brain-derived acetylcholine may also play important role in rhythmic gene expression that influences insulin signaling.[56]

Food intake

Epidemiologic studies have generally not found strong associations between total energy intake and habitual sleep duration.[57,58] However, accurate assessment of diet at a population level is difficult. Several laboratory studies have examined acute effects on eating behavior as a result of reduced sleep. St-Onge and colleagues[59] found that otherwise healthy adults with sleep restricted to 5 hours consumed an extra ~2100 kJ/d. Similarly, Markwald and colleagues[53] found that individuals with sleep restricted to 4 hours consumed an extra ~2100 kJ/d; this study examined timing of food intake and showed that the extra energy consumption occurred at night (with slightly less energy consumption in the morning). These findings were replicated and expanded by Spaeth and colleagues,[60] who similarly examined dietary intake among individuals restricted to 4 hours, over the course of 5 nights.