Invited Commentary: Understanding the Role of Sleep

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Chronic sleep deprivation is increasingly entertained as a novel risk factor for obesity. However, the vast majority of studies on this topic have relied on unvalidated subjective measures of habitual sleep habits. The accompanying paper by Lauderdale et al. (Am J Epidemiol. 2009;170(7):805–813) presents the first longitudinal analysis of the relation between sleep duration and weight change by using an objective assessment of sleep. The lack of evidence for an association in this work suggests that the absolute time slept may not be important for weight regulation but raises questions as to what self-reported sleep duration is measuring. One intriguing possibility is that self-reported sleep may reflect the time spent in deeper stages of sleep, which physiologic studies suggest may be more relevant from a metabolic standpoint. Further research into the relation between sleep quantity and quality relative to obesity by use of more refined measures of sleep is needed to identify which, if any, aspects of sleep are important in weight homeostasis.

Abbreviation: CARDIA, Coronary Artery Risk Development in Young Adults.

Although we spend one third of our lives asleep, the function of sleep continues to elude scientists. Despite the obvious survival disadvantage of a reduced awareness of the environment, the fact that sleep is conserved across a wide range of animal species indicates that sleep must play a fundamental role in health. Epidemiologic studies suggest that chronic sleep deprivation predisposes to mortality, heart disease, diabetes, and most recently obesity. Over a dozen cross-sectional studies have reported an association between short sleep duration and obesity across a wide range of cultures, ethnic groups, and age groups, suggesting a consistent and generalizable association (1). Several longitudinal studies have also demonstrated that reduced sleep predicts an increased rate of weight gain, further increasing interest in sleep curtailment as a novel risk factor for obesity (2, 3).

However, the mechanisms by which reductions in sleep might impact weight remain unknown. Although several studies have identified sleep-related differences in appetite-regulating hormones such as leptin and ghrelin (4, 5), the clinical impact of these changes is unclear, as an increase in caloric intake has not been clearly demonstrated with sleep restriction (6). Similarly, reduced sleep has not been associated with reductions in energy expenditure (6, 7).

A weakness of much of the literature on this topic has been a reliance on self-reported measures of sleep duration. Although individuals demonstrate some ability to assess the amount of sleep they obtained on the night prior to questioning, it is unclear to what extent one can integrate nocturnal sleep with daytime naps and then average habits over multiple nights. This is particularly worrisome in working adults, where substantial variability exists between work nights and weekend nights. In addition, with up to 10% of the workforce involved in shift work, the day-to-day variability in job shift leads to large changes in sleep habits. The most often cited study supporting the use of self-reported assessments of sleep duration identified a moderate correlation with objective measurements of sleep (8). However, this study was performed in a highly selected cohort of blind subjects, raising concerns for generalizability. Of the longitudinal studies identifying an association between sleep duration and weight gain, only the Nurses’ Health Study has attempted to validate the measure of sleep used (9). A single
question on usual sleep duration demonstrated good correlation with the mean obtained from 1 week of sleep diaries. However, this validation study was done in a retired age group with fairly stable sleep habits. In addition, the sleep diaries used as the “gold standard” in that work were also based on subjective recall.

Thus, the analysis by Lauderdale et al. (10) in this issue of the Journal, which presents the first longitudinal analysis of objectively measured sleep duration and weight change, represents a major step forward for the field. Sleep duration was assessed by averaging results from 6 days of actigraphy, which utilizes a wrist accelerometer to detect movement. The amount of motion is used to infer sleep-wake status. Normal individuals, actigraphy correlates well with electroencephalographic measures of sleep, with the advantage that the recording device does not interfere with sleep and can easily record over multiple nights (11).

In agreement with results from prior studies, those from Lauderdale et al. (10) found a fairly robust cross-sectional association in a subgroup of the Coronary Artery Risk Development in Young Adults (CARDIA) Study cohort between short sleep and greater body mass index. Reduced sleep as assessed by actigraphy has been previously associated with increased body mass index in at least 3 cohorts of older individuals—the Study of Osteoporosis, the Male Study of Osteoporosis, and the Rotterdam studies (12, 13). The CARDIA Study now extends these findings to a middle-aged population.

However, the association appeared to be present solely among those reporting snoring, a symptom commonly associated with obstructive sleep apnea. Because obesity is a strong risk factor for sleep apnea and sleep apnea is characterized by disruption of sleep, these findings suggest that the cross-sectional relation between short sleep and obesity may be due to the effects of obesity on sleep rather than vice versa. In support of this causal model, Lauderdale et al. (10) found no association between sleep duration and weight change in the longitudinal analysis. However, unexpectedly, they did identify snoring as a predictor of weight gain. The significance of this finding is unclear. Although obstructive sleep apnea has been associated with elevations in leptin levels and greater weight gain in a retrospective analysis (14), no effect on weight has been found with sleep apnea treatment (15, 16). Further studies with more accurate assessments of apnea are clearly needed to understand whether the relation between obesity and obstructive sleep apnea is bidirectional.

The CARDIA Study results raise another important question: If the association between self-reported sleep and weight gain is not due to an effect of the actual time slept, then what is the explanation for this association? Some have argued that self-reported habitual sleep may represent an overall sense of well-being, with deviations from the norm representing distress of some sort, whether it be psychological, physical, or socioeconomic (17). Another possibility is that self-reported sleep may be a measure of the amount of “quality sleep,” however that may be defined. For example, quality sleep may reflect deeper stages of sleep, such as slow wave sleep, which appears to be particularly relevant for metabolic homeostasis. This stage of sleep is associated with a surge in growth hormone secretion, decline in corticotropic activity, and reductions in sympathetic tone. An experimental paradigm where slow wave sleep was suppressed without changing absolute sleep duration was found to worsen insulin sensitivity (18). In the Male Study of Osteoporosis cohort, a reduced proportion of slow wave sleep was associated with greater body mass index independent of total sleep time (19). Consolidated sleep may be another type of high quality sleep. In the Rotterdam Study where actigraphy was used to assess sleep, the degree of sleep fragmentation was a stronger predictor of adiposity than reduced sleep duration (13). The measure of sleep disruption used by the CARDIA Study investigators also appeared to be more strongly associated with body mass index than total sleep time. Finally, the timing of sleep may be important. Recent work simulating shift work suggests that ensuring sleep is obtained at the right point in one’s circadian rhythm may be as important for metabolism as the amount of sleep obtained (20).

Further understanding of the most relevant sleep-related exposure will be vital to understand how best to measure sleep. If epidemiologists are truly interested in studying the potential role of sleep with regard to health, it is time to move beyond unvalidated questions and to begin to leverage available technologies such as actigraphy to fully characterize sleep exposures, including not only validated measures of sleep duration but also the type of sleep obtained, the timing of that sleep relative to circadian rhythms, and the presence and severity of any sleep disorders. Until such complete assessments occur, we will continue to remain in the dark about the role of sleep.

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REFERENCES


