COMMENTARY

# Obesity and Sleep: A Bidirectional Association?

Commentary on Theorell-Haglöw et al. Associations between short sleep duration and central obesity in women. SLEEP 2010;33:593-598.

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THE ASSOCIATION BETWEEN SLEEP DISORDERS AND OBESITY HAS BEEN RECOGNIZED FOR MANY YEARS. FROM THE FIRST DESCRIPTION OF JOE THE "Fat Boy" who fell asleep in any situation at any time of day (The Pickwick Papers by Charles Dickens), to the large sophisticated studies in clinical and general populations, the association of sleep apnea and obesity has proven to be one of the most well-established facts in the sleep medicine literature. In 2004-2005, the field was jolted by a series of publications on the association of short sleep duration and obesity. Several epidemiologic studies showed a consistent association between self-reported sleep duration and body mass index (BMI),[1-2] whereas experimental laboratory studies showed that curtailment of sleep in healthy subjects leads to increased appetite and reduction of leptin, a hormone that suppresses appetite.[3] The message from these novel findings was simple and exciting, "Sleep more and you will lose weight." This message also had a significant public health impact, given the epidemic of obesity that Western countries have been experiencing since at least the 1980s.

Most of these population studies were based on self-reported sleep duration. To better understand the nature of this association, we examined who among obese individuals report short sleep and what their clinical profiles and characteristics are[4] using a large Penn State Cohort. In our study, consistent with most previous reports, obese individuals reported short sleep duration. In addition, they reported a higher incidence of subjective sleep disturbances (47.4% vs 25.5%, $P < 0.01$) and scored higher for chronic emotional stress than did nonobese subjects. Notably, self-reported sleep duration did not differ between obese and nonobese individuals without subjective sleep disturbances, whereas obese individuals who had sleep complaints scored higher in chronic emotional stress, compared with obese individuals who did not have sleep complaints. The shortest sleep duration was reported by the obese subjects with insomnia (5.9 h), followed by obese individuals with excessive daytime sleepiness (6.3 h) or sleep difficulty (6.6 h). The effect of chronic emotional stress was stronger than that of the BMI on the reported sleep duration, with a synergistic effect between the 2 factors. We concluded that short sleep duration is reported by obese individuals who are unhappy with their sleep and are chronically stressed.

Notably, at the same time, other large epidemiologic studies suggested that self-reported short sleep is influenced by social stressors, such as socioeconomic and minority status,[5] and unhealthy behaviors (i.e., cigarette smoking, consuming 4 or more alcoholic drinks per day, and lack of physical activity).[6] This raised the possibility that self-reported short sleep duration is a marker of sleep complaints and chronic psychosocial stress, which should be the target of our preventative and therapeutic strategies.[7]

Since self-reported sleep duration appears to be influenced by many factors, the interests of several investigators naturally shifted toward examining the association of objective sleep duration and BMI. The first 3 studies found no association or a very weak association between objective sleep duration and BMI.[1,4,8] Two more recent studies using actigraphy reported an association between sleep duration and obesity in elderly community samples; however, the results were weakened after controlling for several confounders.[9-10] The study by Theorell-Haglow and colleagues in this issue of *SLEEP*,[11] is the first using full-night polysomnography to demonstrate a significant inverse association between sleep duration and waist circumference, a marker of central obesity in 400 middle-aged women.[11] Furthermore, duration of slow wave sleep and rapid eye movement sleep were both inversely related to abdominal obesity. These results are significant because they are based on a population sample, using objective sleep measures, and the association with abdominal obesity is very important given the strong evidence linking this type of obesity with cardiometabolic morbidity and mortality. A limitation in the Theorell-Haglow et al. study[11] is that there are no data on the potentially modifying effect of stress and sleep complaints. Interestingly, we have observed that objective sleep abnormalities in obese individuals are strongly associated with emotional stress, whereas, in the absence of sleep complaints or stress, sleep and its structure are not different from those of nonobese control subjects.[7] Furthermore, the results of the study by Theorell-Haglow and colleagues are cross-sectional and do not allow any inference about causality. In fact, in 1994 and 1998[12-13] we reported that the polysomnographically recorded sleep of morbidly obese patients was affected in a very similar way to that reported by Theorell-Haglow et al.[11] Again, this study was cross-sectional, and the direction of the association could not be inferred.

Although cross-sectional studies cannot answer the question about the direction of the association between sleep duration and obesity, longitudinal and intervention studies may shed

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some light. A recent study using actigraphy, cross-sectionally confirmed the previously noted association between short sleep and BMI; however, it failed to show a prospective association between baseline objective sleep duration and development of obesity after 5 years.[14] Furthermore, correction of objective sleep and its structure by the use of continuous positive airway pressure failed to improve weight in a clinical sample of men and women with apnea who were followed for 1 year.[15] In fact, women and those with higher continuous positive airway pressure compliance gained weight! Similar findings were reported in 2006 by Quan in Washington, DC, at the National Sleep Foundation meeting.[16] Clearly, these first findings do not support a simplistic model (i.e., sleep more, increase your slow-wave sleep or rapid eye movement sleep, and you will lose weight).

Do these early findings suggest that objective measures are irrelevant to our health? Recent studies have shown that, in chronic insomnia or poor sleep, objective sleep duration is a strong predictor of cardiometabolic morbidity (e.g., hypertension, diabetes).[17-18] Given that about 50% of obese people complain about the quality of their sleep, it is possible that, in these individuals, objective short or disturbed sleep may be a very useful predictor of cardiometabolic morbidity, which is increased in the obese.

We do not have any doubt that sleep and obesity are strongly connected. The nature and the direction of this association requires further studies—longitudinal, interventional, or both—and using measures from multiple domains, including sleep (both objective and subjective), stress (physiologic, emotional, and social), and mental and physical health. In the meantime, the credibility of our field will be better served if we become comfortable with the suggestion that the obesity epidemic requires a multidimensional approach—that is, stress management, reduction of risky behaviors, promotion of healthy lifestyle at both an individual and a community level, and, of course, better quality and, in some instances, greater quantity of sleep.

## DISCLOSURE STATEMENT

The authors have indicated no financial conflicts of interest.

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