Overweight and Obese Patients in a Primary Care Population Report Less Sleep Than Patients With a Normal Body Mass Index

Robert D. Vorona, MD; Maria P. Winn, MSN, FNP; Teresa W. Babineau, MD; Benjamin P. Eng, MD; Howard R. Feldman, MD; J. Catesby Ware, PhD

Background: Insufficient sleep and obesity are common in the United States. Restricted sleep causes important neurocognitive changes, including excessive daytime sleepiness and altered mood. This may result in work-related injuries and automotive crashes. Evidence links sleep loss to hormonal changes that could result in obesity. This article examines the association between restricted sleep and obesity in a heterogeneous adult primary care population.

Methods: A total of 1001 patients from 4 primary care practices participated in this prospective study. Patients completed a questionnaire administered by a nurse or study coordinator concerning demographics, medical problems, sleep habits, and sleep disorders. Professional staff measured height and weight in the office. The relationship between body mass index (BMI) and reported total sleep time per 24 hours was analyzed after categorizing patients according to their BMI (calculated as weight in kilograms divided by the square of height in meters) as being of normal weight (<25), overweight (25-29.9), obese (30-39.9), or extremely obese (≥40).

Results: Analyzable forms from 924 patients aged between 18 and 91 years indicated that (1) the mean BMI was 30; (2) women slept more than men; (3) overweight and obese patients slept less than patients with a normal BMI (patients reported less sleep in a nearly linear relationship from the normal through the obese group); and (4) this trend of decreasing sleep time was reversed in the extremely obese patients.

Conclusions: This study found that reduced amounts of sleep are associated with overweight and obese status. Interventions manipulating total sleep time could elucidate a cause-and-effect relationship between insufficient sleep and obesity.

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NSUFFICIENT SLEEP CAUSES IMPORTANT neurocognitive changes such as excessive daytime sleepiness, altered mood, and increased risk of work-related injury and automotive crashes. Dawson and Reid1 equated performance after 24 hours without sleep to performance with a blood alcohol level of 0.10%. In the United States, insufficient sleep is a common phenomenon. For example, the National Sleep Foundation 2000 Omnibus “Sleep in America” poll2 documented that the average adult admitted to 7 hours of sleep per night. While estimates that the average American slept about 9 hours per night a century ago.3

See also pages 15, 35, and 42

In concert with declining sleep times there has been an increase in the prevalence of both obesity and severe obesity. A comparison of studies conducted from 1988 to 1994 and from 1999 to 2000 determined that the prevalence of obesity has increased from 23% to 31% and the prevalence of severe obesity from 3% to 5% between the 2 periods.4 No sex or racial group is exempt from this significant health problem. Recent data also indicate an increasing prevalence of obesity among children.5

Two recent studies from Japan have explored the relationship between insufficient sleep and obesity. In 8274 children aged from 6 to 7 years, Sekine et al6 found an inverse relationship between hours of sleep and risk of childhood obesity (defined as body mass index [BMI], calculated as weight in kilograms divided by the height in meters).
Body mass index (calculated as weight in kilograms divided by the square of height in meters) did not significantly differ between men and women.

Recent findings suggest that endocrine changes may mediate this relationship. Changes in the consequences of inadequate sleep time.

The questionnaire inquired about basic demographics; the presence, frequency, and duration of naps; bed time, wake time, and total estimated sleep time per 24 hours; general medical population. Additional information linking these 2 important problems might spur rethinking the consequences of inadequate sleep time.

METHODS

PARTICIPANTS

A total of 1001 individuals aged from 18 to 91 years were recruited from 4 primary care practices in the southeastern portion of Virginia. Enthusiasm and close attention by the nursing staff and study coordinators allowed the investigative team to recruit more than 95% of the patients approached for the study. They came from 2 family practice residency training centers at Eastern Virginia Medical School, 1 internal medicine practice at Eastern Virginia Medical School, and 1 private family practice. The institutional review board of the Eastern Virginia Medical School approved the study.

PROCEDURES

As part 1 of the patients’ regularly scheduled visits to their primary care clinician the professional staff obtained characteristics such as height and weight. Either the patients completed the study questionnaire and it was immediately reviewed by the research staff or the nursing staff completed it while interviewing the patients. The staff attempted to select consecutive patients. The questionnaire inquired about Basic demographics; the presence, frequency, and duration of naps; bed time, wake time, and total estimated sleep time per 24 hours; general medical problems; diagnosed sleep disorders; and sleep hygiene issues, which included the use of caffeine, tobacco, alcohol, and weight loss products. Questionnaire data were entered in a single database for analysis. Of the 1001 questionnaires administered, 929 had the key items completed (ie, age, sex, weight, height, and total sleep time per 24 hours [TST] during the workweek and weekend). A preliminary review identified 5 weekday sleep times that were statistical outliers (105, 870, 900, 960, and 1200 minutes). As these values suggested errors in completion, transcription, or understanding of the questions, the patients who reported these sleep times were dropped from the analysis, leaving 924 participants. Weekday but not weekend sleep time was recorded for 3 participants, for whom we used the weekday sleep values also for the weekend. In the analysis, total weekday and weekend sleep times were combined with appropriate weighting for the number of days.

We analyzed the relationship between BMI and TST by categorizing participants into 4 clinically recognizable BMI groups: normal, overweight, obese, and extremely obese. These groups are similar to those established by the National Heart, Lung and Blood Institute as of April 2004 (available at: http://www.nhlbi.nih.gov/guidelines/obesity/ob_tbl2.htm). The 4 groups were composed of persons with BMIs of 17 to 24 (n = 203), 25 to 29 (n = 288), 30 to 39 (n = 339), and 40 to 85 (n = 94), respectively. Total sleep time was examined in an analysis of variance using BMI grouping and sex as between-subject variables.

STUDY POPULATION

The mean age, weight, height, and BMI of the participants are presented in Table 1. Most participants were obese and middle-aged (ages ranged from 18 to 91 years). As would be expected in individuals recruited from primary care practices, medical problems were often present (hypertension was particularly prevalent) (Table 2). The proportions of patients self-reporting insomnia, narcolepsy, obstructive sleep apnea syndrome (OSAS), periodic limb movements of sleep and restless legs syndrome are shown in Table 3. Patients most commonly reported insomnia. Total sleep times for those with and

### Table 1. Age, Weight, Height, and Body Mass Index (BMI) of Participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. of Participants</th>
<th>Age, y</th>
<th>Weight, kg</th>
<th>Height, cm</th>
<th>BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men*</td>
<td>326</td>
<td>50 ± 15</td>
<td>92.2 ± 20.0</td>
<td>172 ± 25</td>
<td>30 ± 6</td>
</tr>
<tr>
<td>Women</td>
<td>598</td>
<td>46 ± 16</td>
<td>80.8 ± 21.3</td>
<td>160 ± 42</td>
<td>30 ± 6</td>
</tr>
<tr>
<td>Total</td>
<td>924</td>
<td>48 ± 16</td>
<td>84.9 ± 21.8</td>
<td>164 ± 37</td>
<td>30 ± 7</td>
</tr>
</tbody>
</table>

*Men were older than women (t922=3.49, P<.001); weighed more (t868=8.07 [unequal variances], P<.001); and were taller (t922=–8.07, P<.001).

Body mass index (calculated as weight in kilograms divided by the square of height in meters) did not significantly differ between men and women.

### Table 2. Participants Reporting Common Medical Disorders

<table>
<thead>
<tr>
<th>Variable</th>
<th>Arthritis</th>
<th>Asthma</th>
<th>CAD</th>
<th>DM</th>
<th>Hypertension</th>
<th>GERD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>88 (27)</td>
<td>32 (10)</td>
<td>44 (14)</td>
<td>68 (21)</td>
<td>137 (42)</td>
<td>65 (20)</td>
</tr>
<tr>
<td>Women</td>
<td>216 (36)</td>
<td>100 (17)</td>
<td>42 (7)</td>
<td>87 (14)</td>
<td>226 (38)</td>
<td>159 (27)</td>
</tr>
<tr>
<td>Total</td>
<td>304 (33)</td>
<td>132 (14)</td>
<td>86 (9)</td>
<td>155 (17)</td>
<td>363 (39)</td>
<td>224 (24)</td>
</tr>
</tbody>
</table>

Abbreviations: CAD, coronary artery disease; DM, diabetes mellitus; GERD, gastroesophageal reflux disease.

The mean age, weight, height, and BMI of the participants are presented in Table 1. Most participants were obese and middle-aged (ages ranged from 18 to 91 years). As would be expected in individuals recruited from primary care practices, medical problems were often present (hypertension was particularly prevalent) (Table 2). The proportions of patients self-reporting insomnia, narcolepsy, obstructive sleep apnea syndrome (OSAS), periodic limb movements of sleep and restless legs syndrome are shown in Table 3. Patients most commonly reported insomnia. Total sleep times for those with and
without specific sleep disorders are presented in Table 4.

Table 3. Participants Reporting Sleep Disorders

<table>
<thead>
<tr>
<th>Variable</th>
<th>Insomnia</th>
<th>Narcolepsy</th>
<th>OSAS</th>
<th>PLMS</th>
<th>RLS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>87 (27)</td>
<td>0</td>
<td>24 (7)</td>
<td>25 (8)</td>
<td>23 (7)</td>
</tr>
<tr>
<td>Women</td>
<td>211 (35)</td>
<td>2 (0.3)</td>
<td>34 (6)</td>
<td>45 (8)</td>
<td>48 (6)</td>
</tr>
<tr>
<td>Total</td>
<td>298 (32)</td>
<td>2 (0.2)</td>
<td>58 (6)</td>
<td>70 (8)</td>
<td>71 (8)</td>
</tr>
</tbody>
</table>

Abbreviations: OSAS, obstructive sleep apnea syndrome; PLMS, periodic limb movements of sleep; RLS, restless legs syndrome.

Table 4. Total Sleep Time (TST) per 24 Hours in Participants With and Without Sleep Disorders*

<table>
<thead>
<tr>
<th>Sleep Disorder</th>
<th>No. of Participants</th>
<th>TST, Mean (SD)</th>
<th>t Test</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>OSAS</td>
<td>Yes</td>
<td>58</td>
<td>495 (110)</td>
<td>2.29</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>865</td>
<td>465 (96)</td>
<td>-0.03</td>
</tr>
<tr>
<td>RLS</td>
<td>Yes</td>
<td>71</td>
<td>467 (114)</td>
<td>-0.03</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>851</td>
<td>467 (96)</td>
<td>0.03</td>
</tr>
<tr>
<td>Narcolepsy</td>
<td>Yes</td>
<td>70</td>
<td>471 (117)</td>
<td>0.30</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>624</td>
<td>472 (96)</td>
<td>0.15</td>
</tr>
</tbody>
</table>

Abbreviations: See Table 3.
*P values were not significant (Bonferroni correction for multiple [5] tests).
†There were unequal variances between groups.

REPORTED TST

The mean ± SD 24-hour TST for the study population was 467 ± 97 minutes. Total sleep time varied with BMI as illustrated in the Figure. Total sleep time decreased as BMI increased, except in the extremely obese group (F3,919 = 3.36, P = .02) (Figure). Patients in the obese group slept less than patients in the overweight group (P = .04), but patients in the overweight group did not sleep significantly less than patients with a normal BMI (P = .31). The difference in total sleep time between patients in the normal weight group (BMI, 22 ± 1.8) and the other patients (BMI, 32 ± 6.6) was 16 minutes per day. Over 7 days, this difference would reach 112 minutes, or 1.86 hours per week. Because 1.86 hours per week is associated with a difference in BMI of 10 between patients in the normal weight group and the other patients, a 1-hour per week difference in total sleep time is equivalent to a mean BMI increase of 5.4.

Because the participants reporting sleep disorders (obstructive sleep apnea syndrome, narcolepsy, insomnia, restless legs syndrome, and periodic limb movements of sleep) may have distorted the relationship between BMI and TST, we repeated the analysis of variance, excluding the 357 patients reporting a sleep disorder. The results, also shown in the Figure, are similar to the results that included individuals with sleep disorders. The normal-weight and overweight groups significantly differed (P = .05), whereas the overweight and obese groups approached being significantly different (P = .07), with the heavier group sleeping less. The obese and extremely obese groups did not differ significantly. Comparing TST for participants with and without specific sleep disorders, the group with OSAS reported sleeping 30 minutes more per 24 hours than the group without OSAS. This difference was not significant when the P value was corrected for multiple (4) tests. Patients identifying insomnia as a problem slept less, but not significantly so (Table 4).

Table 5 displays the relationship between the medical disorders reported by the participants and TST. Even when uncorrected for repeated tests, there was no significant difference in TST between participants with and without medical disorders.

Sleep hygiene issues may affect TST. Therefore, the relationships among shift work, smoking, caffeine, weight loss products, and TST are displayed in Table 6. Nightshift work was associated with 42 minutes less TST (t183 = 3.162, P = .002). Evening shift work, smoking, caffeine, and weight loss products did not affect TST.

Because healthy men and women have been reported to have different total sleep times, we examined TST by sex. Men reported less sleep than women (450 ± 95 minutes vs 477 ± 97 minutes; F1.927 = 6.83, P = .009).
There was no interaction between sex and BMI category ($F_{3,917}=0.72$).

This prospective study examining reported TST in relation to obesity has 4 noteworthy findings. First, patients of the 4 southside Virginia primary care practices where this study was conducted had a mean BMI of 30. Per National Institutes of Health criteria, the average patient in these 4 practices was obese. Unsurprisingly, the study participants had weight-associated medical problems, such as diabetes mellitus, hypertension, arthritis, and gastroesophageal reflux disease. Within this general medical population, study participants with these problems did not report different TSTs. Although the participants may have underestimated or overestimated the occurrence of their medical problems, the questionnaires were completed in their physician's office, most often by a professional with the medical records at hand. Such an environment should contribute to more accurate data acquisition than patient-completed questionnaires done at home. And although the questionnaire's list of medical problems that could interfere with sleep was not exhaustive (for example, there was no question concerning chronic pain), arthritis, a potential proxy for pain and hence perturbed sleep, was not associated with altered TSTs.

Second, reported TST varied neither with the sleep hygiene factors of smoking, drinking coffee, and using weight loss products, nor with evening shift work. Third, 3 factors affected reported TST: being a night-shift worker (as has been previously shown), a man (across all BMI categories), and obese.

Finally, an increasing BMI—from normal, to overweight, to obese—was associated with a decreasing TST. This association was noted both in the total population of participants and in participants who reported no sleep disorders. The association between obesity and decreasing TST in this study supports earlier findings concerning Japanese adults and children.6,7

Restricted sleep and obesity could be associated for several reasons. First, recent investigations document that insufficient sleep causes “peripheral effects” that may potentiate obesity. Spiegel et al8 reported metabolic and hormonal consequences of acute sleep restriction. Young healthy men limited to 4 hours of sleep for 6 consecutive nights experienced reduced glucose tolerance, reduced glucose effectiveness, and acute insulin response to glucose. The same study demonstrated an increase in both sympathovagal balance and evening and nocturnal cortisol levels; and it also demonstrated lower rises in nocturnal and 24-hour mean thyrotropin concentrations. Spiegel et al compared these perturbations to changes that occur in aging or in gestational diabetes. In addition, the authors hypothesized that, over time, worsened carbohydrate handling and increased sympathetic activity might
result in such problems as hypertension and obesity. Moreover, they demonstrated that sleep restriction in men causes changes in growth hormone secretion, which could lead to suboptimal handling of glucose. Finally, they posited that the actual timing of sleep restriction, in addition to sleep restriction itself, affected growth hormone secretion patterns and hence glucose control. Others, eg, Gonzalez-Ortiz et al,13 determined that 24 hours of sleep deprivation decreased insulin sensitivity but did not alter cortisol levels; and Lusardi et al12 determined that sleep deprivation in hypertensive persons was associated with rises in blood pressure, heart rate, and nocturnal levels of norepinephrine.

Furthermore, sleep restriction reduces the adipocyte-secreted and appetite-regulating hormone leptin, and therefore increases appetite.10,15 Thus leptin, in part, may mediate the sleep-obesity relationship. Another reason for the sleep-obesity relationship may be very simple: increased time awake means increased time to eat.

In contrast to the overweight and obese patients, the extremely obese patients in our study did not demonstrate a further decline in TST. Instead, they slept more than the obese patients. Why, then, might the extremely obese not demonstrate the shortest sleep times?

In a study by Vgontzas et al16 obese individuals without a sleep breathing disorder who reported increased subjective sleepiness also had objective signs of sleepiness such as shorter sleep latency during daytime naps. This finding suggests, as the authors concluded, that obesity itself may lead to excessive daytime sleepiness. Certain cytokines (eg, tumor necrosis factor α and interleukin 6) were shown to have sleep-inducing effects,17 and there is evidence that levels of soporific cytokines may be increased in obese persons. For example, BMI and levels of interleukin 6 correlate in humans,17 and tumor necrosis factor α levels are elevated in obese animals.18 It is possible that our extremely obese study participants did not have a further decline in TST owing to increased levels of these cytokines.

Although not statistically significant, participants with OSAS reported a mean of 30 more minutes of sleep per night than the group without OSAS. It seems reasonable to postulate that the extremely obese group may have included participants with unreported OSAS,19 thus resulting in mean increased TST for this group. Finally, the presumed heterogeneity of disorders in patients from the extremely obese group seeing their primary care physicians, and presumably taking a wide range of medications, may have distorted the sleep-obesity relationship. Collecting data from a healthier population would help answer this question.

Nevertheless, despite the self-reported sleep data, which can be more variable and less accurate than objective measures of sleep, we found a relationship between TST and BMI. Therefore, studies using tools such as sleep logs, actigraphy, or polysomnography may show an even stronger relationship. We also believe that further studies would do well to evaluate patient medications more completely for their role in sleep duration.

This study adds to the literature by demonstrating peripheral effects of sleep deficit. Among the study’s strengths are a large heterogeneous group recruited from 4 different practice sites. In addition, previous work demonstrating a physiologic basis for linking insufficient sleep and an increased risk of obesity provided a framework for this study’s hypothesis. Americans experience insufficient sleep and corpulent bodies. Clinicians are aware of the burden of obesity on patients. Certainly, factors other than sleep limitation (eg, diet and inactivity) contribute to obesity in the United States. We also admit that some medical problems associated with obesity cause discomfort, and that discomfort, by preventing patients from sleeping, may increase time for caloric intake. Follow-up studies that include measures of caloric intake would be helpful. Despite the mentioned caveats, the contribution of insufficient sleep to obesity merits further investigation. If insufficient sleep were to be definitively determined to contribute to obesity, aesthetic and health considerations might lead many Americans to pay more attention to the benefits of adequate sleep.

Finally, should adequate sleep be recommended in weight loss programs, along with exercise and proper diet? Can the statistical relationship shown in this study translate into meaningful clinical effect? Our findings suggest that major extensions of sleep time may not be necessary, as an extra 20 minutes of sleep per night seems to be associated with a lower BMI. We caution that this study does not establish a cause-and-effect relationship between restricted sleep and obesity. Investigations demonstrating success in weight loss via extensions of sleep would help greatly to establish such a relationship.

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Announcement

Online Submission and Peer Review System to Be Available in February 2005. The Archives of Internal Medicine editorial office will be introducing an online manuscript submission and peer review system developed by ejournalPress that will serve the needs of authors, reviewers, and editors. The new system is scheduled to go live on February 14. See http://www.archinternmed.com for more detailed information.