A Prospective Study of Self-Reported Sleep Duration and Incident Diabetes in Women

NAJIB T. AYAS, MD[1,2]
DAVID P. WHITE, MD[1,2]
WAEL K. AL-DELAIMY, MD, PHD[3]
JOANN E. MANSON, MD, DRPH[2,4,5,6]
MEIR J. STAMPFER, MD, DRPH[3,4,6]
FRANK E. SPEIZER, MD[2,6]
SANJAY PATEL, MD[1,2]
FRANK B. HU, MD, PHD[3,4,6]

Short-term sleep restriction results in impaired glucose tolerance. To test whether habitually short sleep duration increases the risk of developing diabetes, we studied a cohort of 70,026 women enrolled in the Nurses Health Study, without diabetes at baseline, and who responded to a question about daily sleep duration in 1986. Subjects were followed until 1996 for the diagnosis of diabetes (1,969 cases). Long and short sleep durations were associated with an increased risk of diabetes diagnosis. The relative risks (RRs) for short (slept ≤5 h per day) and long (slept ≥9 h per day) sleepers were 1.57 (95% CI 1.28–1.92) and 1.47 (1.19–1.80), respectively. After adjustment for BMI and a variety of confounders, the RR was not significantly increased for short sleepers (1.18 [0.96–1.44]) but remained modestly increased for long sleepers (1.29 [1.05–1.59]). We then performed a similar analysis using only symptomatic cases (n = 1,187). Adjusted RRs for symptomatic diabetes were modestly elevated in both short (1.34 [1.04–1.72]) and long (1.35 [1.04–1.75]) sleepers. Our data suggest that the association between a reduced self-reported sleep duration and diabetes diagnosis could be due to confounding by BMI, or sleep restriction may mediate its effects on diabetes through weight gain. Sleep restriction may be an independent risk factor for developing symptomatic diabetes.

Diabetes Care 26:380–384, 2003

Chronic sleep loss is very common in today's society. Even though sleep professionals and the National Sleep Foundation recommend 8 h of sleep per night, American adults only sleep an average of 6.85 h per night (1). Furthermore, only 37% report obtaining ≥8 hours of sleep per night, and 31% of adults report sleeping <6 h per night. The amount of sleep individuals obtain also is steadily decreasing over time. Thirty years ago, adults slept ~7.68 h per night; 80 years ago, they slept even longer (8.77 h reported by college-aged adults) (2,3). The cause of this sleep loss is multifactorial. About 45% of adults report that they sleep less to get more work done, 43% stay up watching television or using the internet, and 22% have insomnia (i.e., report having difficulty falling asleep). Because of these figures, many have suggested that we live in a sleep-deprived society. (4)

The health effects of sleep restriction are unclear. However, a recent study demonstrated that sleep deprivation can adversely affect endocrine function. Spiegel et al. (5) limited 11 young men to 4 h of sleep per night for 6 nights followed by 6 days of recovery sleep (10 h per night). Despite the short duration of partial sleep deprivation, the subjects demonstrated impaired glucose tolerance during sleep deprivation compared with their recovery period.

In this study, we assessed the relationship between self-reported sleep duration and the diagnosis of diabetes during 10 years of follow-up among women enrolled in the Nurses Health Study. We hypothesized that sustained short sleep duration could lead to the development of overt clinical diabetes.

RESEARCH DESIGN AND METHODS

Study population

The Nurses Health Study cohort was established in 1976 when 121,700 female married registered nurses, aged 30–55 years and residing in 11 large U.S. states, completed a mailed questionnaire on their medical history and lifestyle. The original sampling strategy for this cohort has been described (6). Every 2 years, follow-up questionnaires have been sent to update information on potential risk factors and to identify newly diagnosed cases of diabetes and other diseases. In 1986, subjects were asked the following question: "Indicate total hours of actual sleep in a 24-h period." (The question did not specify the time frame.) The nurses were asked to choose one of the following options: 5 h or less, 6 h, 7 h, 8 h, 9 h, 10 h, or 11+ hours. Between 1986 and 1996, the incidence rate of diabetes diagnosis was assessed in the 70,026 women who answered this question and did not have a diagnosis of diabetes or cancer in 1986 (the baseline year for these analyses).

Diagnosis of diabetes

A supplementary questionnaire regarding symptoms, diagnostic tests, and hypoglycemic therapy was mailed to women who indicated on any biennial questionnaire that they had been diagnosed with diabetes. A case of diabetes was considered confirmed if at least one of the following was reported on the supplementary questionnaire: *1*) one or more classic symptoms (excessive thirst, coma, polyuria,

From the [1]Division of Sleep Medicine, Department of Medicine, Brigham and Women's Hospital, Boston; [2]Harvard Medical School, Boston; the [3]Department of Nutrition, Harvard School of Public Health, Boston; the [4]Department of Epidemiology, Harvard School of Public Health, Boston; the [5]Division of Preventive Medicine, Department of Medicine, Brigham and Women's Hospital, Boston; and the [6]Channing Laboratory, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts.

Address correspondence and reprint requests to Frank B. Hu, MD, PhD, Dept. of Nutrition, Harvard School of Public Health, 665 Huntington Ave., Boston, MA 02115. E-mail: frank.hu@channing.harvard.edu.

Received for publication 16 July 2002 and accepted in revised form 29 October 2002.

Abbreviations: RR, relative risk.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

Table 1—*Baseline characteristics and risk factors for diabetes according to self-reported sleep duration in 1986*

	Hours of sleep per day				
	≤5	6	7	8	9+
Mean					
Age (years)	52.8	52.2	51.9	52.3	52.6
BMI (kg/m^2)	25.2	24.9	24.4	24.5	24.8
Alcohol cconsumption (g/day)	5.0	5.8	6.3	6.9	8.1
Physical activity (METs/week)	14.3	14.2	14.6	14.1	12.2
% of women					
History of hypercholesterolemia	13.0	10.9	10.4	11.4	11.5
History of hypertension	27.5	23.8	21.1	22.5	25.9
Current smoking	23.5	23.7	20.6	19.6	22.6
Family history of diabetes	17.3	16.8	15.9	16.0	16.0
Regular snoring	10.0	9.6	8.2	9.3	11.5
Night shift work >5 years*	27.6	19.5	14.9	14.1	15.7
Current postmenopausal hormone use	16.6	21.2	23.4	22.7	22.4

*Reported in 1988. METs = metabolic equivalent.

weight loss, hunger, pruritis) plus a fasting plasma glucose of ≥140 mg/dl (7.8 mmol/l) or a random plasma glucose of ≥200 mg/dl (11.1 mmol/l); 2) at least two elevated plasma glucose concentrations on different occasions (fasting ≥140 mg/dl [7.8 mmol/l] or a random plasma glucose of at least 200 mg/dl [11.1 mmol/l] and/or concentration ≥200 mg/dl after ≥2 h on oral glucose tolerance testing) in the absence of symptoms; or 3) treatment with hypoglycemic medication (insulin or oral hypoglycemic agent). Our criteria for diabetes classification are consistent with those proposed by the National Diabetes Data Group (7). The validity of this questionnaire has been verified in a subsample of this study population. Among a random sample of 84 women classified by the questionnaire as having type 2 diabetes, 71 gave permission for their medical records to be reviewed, and records were available for 62. An endocrinologist (J.E.M.), blinded to the information reported on the supplementary questionnaire, reviewed the records according to National Diabetes Data Group criteria. The diagnosis of type 2 diabetes was confirmed in 61 of 62 (98%) of the women (8).

We first performed the analysis using total cases of incident diabetes diagnosis. To address the possibility that self-reported sleep duration may be associated with developing more severe forms of diabetes, we then performed an analysis restricted to cases reporting at least one of the following symptoms of diabetes at diagnosis: pruritis, coma, urinary frequency, hunger, weight loss, and thirst.

Statistical analysis

Person-time for each exposure category (sleep duration of ≤5 h per night, 6 h per night, 7 h per night, 8 h per night, or ≥9 h per night) was accumulated, and incidence rates were calculated by dividing the number of events by person-time of follow-up in each category. Because of the small number of subjects, we combined the subjects from the 9, 10, and ≥11 hours of sleep into one group. The relative risk (RR) was computed as the rate in a specific category of exposure divided by that in the reference category with adjustment for age. We chose a reference category of 8 h per night for two reasons. First, 8 h is conventionally considered to be the appropriate duration of sleep. Second, in a previous study using this cohort, individuals reporting 8 h of sleep per night had the least risk of developing coronary artery disease (9).

In multivariate analyses using pooled logistic regression (10), we simultaneously included age (5-year interval), smoking status (never, past, current smoking of 1–14, 15–24, and ≥25 cigarettes per day), hypertension, alcohol consumption (0, 1–4, 5–14, ≥15 g/day), physical activity (weekly energy expenditure in metabolic equivalent hours), menopausal status (premenopausal, postmenopausal without hormone replacement, postmenopausal with past hormone replacement, postmenopausal with current hormone replacement), depressed mood from 1992 (depression was defined as an Short Form-36 mental health index of ≤52), family history of diabetes, and a history of hypercholesterolemia. We felt it necessary to control for all these factors given that any of them (e.g., depression) can be associated with sleep duration and/or diabetes. Thus, all could be potential confounders in our study. Because shift-working may be associated with poorer health and short sleep duration (11), we controlled for shift-working using the duration of rotating night shift work reported in 1988 (four categories: 0, 1–5, 6–14, ≥15 years). We also controlled for snoring (reported in 1986), as this symptom has been shown to be associated with incident diabetes diagnosis in this cohort (12).

In the primary analysis, we did not control for BMI because sleep restriction decreases leptin levels, and may thus be associated with increased weight gain (13). That is, weight gain may be an intermediate mechanism whereby short sleep leads to the development of diabetes. In secondary analyses, we adjusted for BMI (eight categories).

RESULTS — Baseline characteristics of the women in the various sleep duration categories are shown in Table 1. At baseline, 4.3% of women reported sleeping ≤5 h a day, 25.5% 6 h, 42% 7 h, 23.6% 8 h, and 4.5% ≥9 h. At baseline, both long and short self-reported sleep durations were associated with an increased prevalence of hypertension and hypercholesterolemia. Women in these sleep categories also tended to be heavier. Regular snoring was slightly more common in those who slept for a longer duration (11.5%). Also, long sleepers consumed more alcohol and exercised less. Nurses who slept less tended to report more shift-working.

The primary analysis was the ascertainment of incident diagnoses of diabetes during the 10-year study period. During this time period, we documented 1,969 incident diagnoses of diabetes. After adjustment for age, both long and short sleep duration were associated with a significantly increased risk of incident diabetes diagnosis (Table 2). For short sleepers (those who slept ≤5 h per night), the RR was 1.57 (95% CI 1.28–1.92), whereas in long sleepers (those who slept ≥9 h per night), the respective RR was 1.47 (1.19–

Table 2—*Relative risks (95% CIs) of incident diabetes according to self-reported sleep duration at baseline*

	Hours of sleep per day				
	≤5	6	7	8	9+
Total diabetes					
n	122	576	731	422	118
Person years	28,608	169,859	281,601	157,904	29,941
Age-adjusted relative risk	1.57 (1.28–1.92)	1.27 (1.12–1.44)	0.98 (0.87–1.11)	1	1.47 (1.19–1.80)
Multivariate model without BMI*	1.29 (1.05–1.58)	1.16 (1.02–1.32)	1.02 (0.91–1.16)	1	1.32 (1.07–1.62)
Multivariate model including BMI (8 categories)*	1.18 (0.96–1.44)	1.10 (0.97–1.25)	1.02 (0.91–1.16)	1	1.29 (1.05–1.59)
Symptomatic diabetes					
Age-adjusted relative risk	1.85 (1.44–2.37)	1.33 (1.13–1.56)	0.97 (0.83–1.13)	1	1.54 (1.18–2.01)
Multivariate model without BMI*	1.52 (1.19–1.96)	1.21 (1.03–1.43)	1.01 (0.86–1.18)	1	1.39 (1.07–1.81)
Multivariate model including BMI (8 categories)*	1.37 (1.07–1.77)	1.13 (0.96–1.34)	1.00 (0.86–1.18)	1	1.36 (1.04–1.73)

*Adjusted for shiftworking (from 1988), hypercholesterolemia, hypertension, smoking, snoring, exercise, alcohol, depression (from 1992), postmenopausal hormone use, and family history of diabetes.

1.80). After adjusting for relevant covariables other than BMI (see Table 2 for list of covariables), the positive associations for diabetes were attenuated but remained statistically significant. Without adjustment for BMI, multivariate RRs for total diabetes were 1.29 (1.05–1.58) for short sleepers and 1.32 (1.07–1.62) for long sleepers. After additional adjustment for BMI, the multivariate RR of diabetes was reduced and no longer significant (1.18 [0.96–1.44]) for short sleepers, but still remained modestly but significantly increased for long sleepers (1.29 [1.05–1.59]).

To assess whether sleep duration was associated with more severe manifestations of diabetes, we performed a similar analysis using only symptomatic cases of diabetes (n = 1,187) (Table 2). Age-adjusted risks for developing symptomatic diabetes were 1.84 (1.43–2.35) in short sleepers and 1.54 (1.19–2.00) in long sleepers. After adjustment for BMI, the relative risks for developing diabetes were modestly elevated in both short (1.34 [1.04–1.72]) and long (1.35 [1.04–1.75]) sleepers.

Stratified analysis was then performed for all diagnoses of diabetes, and also for symptomatic cases. Stratification was done by BMI (three categories: ≤25, 25–30, ≥30 kg/m^2), shift-working (three categories: 0–5, 6–14, >15 years), and snoring (three categories: regularly, occasionally, and never). Results were not substantially different in each of the strata.

To address the possibility that subclinical diabetes may have led to sleep complaints (i.e., reverse causation), we conducted additional analyses by excluding cases of type 2 diabetes that were diagnosed during the first 2 years of follow-up (1,621 cases). The multivariate RRs (excluding BMI) across the various sleep durations (≤5, 6, 7, or ≥9 h) were 1.50, 1.25, 0.97, and 1.40, respectively; these values were very similar to the values obtained without the lag analysis. That is, exclusion of these cases did not change the RRs substantially, suggesting that reverse causation was not a likely explanation for our findings. When we performed a lag analysis considering only symptomatic cases of diabetes (n = 965), the adjusted RRs (including control for BMI) followed a trend similar to that of our primary analysis (1.28, 1.13, 0.97, and 1.30 for ≤5, 6, 7, and ≥9 h, respectively).

DISCUSSION — We observed a modest but significant positive association between self-reported sleep duration and incident diagnosis of diabetes. Both short and long self-reported sleep durations were associated with an increased risk of developing diabetes. For short sleepers, after controlling for BMI, the relative risk was attenuated and no longer significant, reflecting a confounding effect of BMI. In long sleepers, a modest but significant positive association between sleep duration and diabetes persisted even after controlling for BMI. When the relationship between self-reported sleep duration and symptomatic diabetes was examined, short sleep duration remained a significant predictor even after controlling for BMI.

Short-term physiologic studies have shown that restriction of sleep results in abnormalities of endocrine function that could predispose to the development of overt diabetes. In a recently published study, Spiegel et al. (5) restricted sleep in 11 healthy young men to 4 h per night for 6 nights and then allowed them to have a sleep recovery period of 6 nights. Despite the short duration of partial sleep deprivation, the subjects in that study demonstrated impaired glucose tolerance, higher evening cortisol levels, increased sympathetic nervous system activity, and a reduction in leptin secretion in the sleep-deprived versus the recovery state (13). Furthermore, in a preliminary study, this same group of investigators compared glucose tolerance and insulin resistance in healthy subjects who regularly slept <6.5 h per night to those who slept 7.5–8.5 h (14). Although the glycemic response to glucose tolerance testing was similar in both groups, the short sleepers had a 50% higher insulin level. These results suggest that habitually short sleep results in a reduction in insulin sensitivity, and therefore, could be a risk factor for the later development of diabetes.

Our study is consistent with these physiologic data. In our study, short self-reported sleep duration was significantly associated with the diagnosis of diabetes until we controlled for BMI. Once BMI was added into the model, the association between diabetes diagnosis and short

sleep was no longer significant. This finding could be explained in a couple of ways. First, short sleep may not be a risk factor for diabetes per se but is instead caused by weight gain. For instance, a high BMI may worsen sleep quality by creating a prediabetic state with increasing urination. Second, sleep restriction may directly lead to the development of diabetes through its effects on weight. Relevant to this hypothesis, leptin (a hormone that suppresses appetite) is reduced in men subjected to short-term sleep deprivation. If chronic self-imposed sleep restriction also leads to reductions in leptin, appetite and weight gain may be increased. This could thus represent a physiologic mechanism whereby sleep restriction may predispose to weight gain and subsequently contribute to the development of diabetes.

When analysis was limited to symptomatic cases of diabetes (considered a more severe diabetes in the analysis), the results were somewhat different. When only symptomatic cases were considered, short sleep duration was associated with an increased risk even after controlling for BMI. One possible explanation of the discrepancy between symptomatic and total cases is that sleep restriction may be associated with more severe forms of diabetes. Another possible explanation is surveillance bias. Participants with restricted sleep may visit physicians less frequently, resulting in an increased risk of presenting as symptomatic as opposed to asymptomatic. We doubt this is a likely explanation because short sleepers actually had more comorbid diseases at baseline, making it more likely they would visit a physician. In addition, all subjects were trained nurses and knowledgeable about health care. For these two reasons, we believe surveillance bias is an unlikely explanation for our findings.

In our study, a long self-reported sleep duration was also associated with an increased risk of developing a diagnosis of diabetes. We speculated on possible explanations for this association. One possibility is that an unrecognized confounder could lead to both diabetes and an increased need for sleep. An example of such a confounder might be obstructive sleep apnea. Sleep apnea is independently associated with decreased glucose tolerance and is known to fragment sleep (15,16). Second, excessive sleep per se could directly lead to an increased risk of diabetes. Currently, however, we know of no plausible physiologic explanation for such a cause-and-effect relationship. Third, long sleep duration may be an early symptom of diabetes, possibly predating its official diagnosis. However, an analysis that excluded cases of diabetes diagnosed within the first two years of reported sleep duration did not yield appreciably different results. Therefore, we also doubt this to be an explanation of our findings. Although the mechanisms underlying the association between long sleep duration and diabetes are not readily explainable, our study is consistent with a recently published article by Kripke et al. (17). In that study, long self-reported sleep duration was also associated with adverse health outcomes; in particular, long sleep (≥8 h per night) was associated with increased all-cause mortality.

Strengths and limitations

Strengths of the current study include the large sample size and the high rate of follow-up. Also, diabetes was assessed prospectively, eliminating possible bias due to retrospective recall that may be present in case-control and cross-sectional analyses.

However, some limitations of this study deserve attention. First, because our "nondiabetic" cohort was not necessarily screened for glucose intolerance, some cases of diabetes may have been undiagnosed. The diagnostic criteria for type 2 diabetes were changed in 1997 such that lower fasting glucose levels (>126 mg/dl) would now be considered diagnostic (18). We used the criteria proposed by the National Diabetes Data Group because all our cases were diagnosed before June 1996 (7). If the new criteria were used, some women in this study classified as nondiabetic would have been reclassified as having diabetes. However, this would not explain our results because, if anything, inclusion of diabetic individuals in the nondiabetic groups would have caused bias toward the null. Second, our subjects were only women, the vast majority Caucasian, and had a similar socioeconomic status. Although the homogeneity in the sample would reduce confounding by these factors, it may reduce the generalizability of our findings to other populations. Currently, however, we have no reason to suspect that men or non-Caucasians would differ in terms of the effects of sleep duration on diabetes. Third, information about sleep duration and other potential risk factors was self-reported by the nurses; the reliability of self-reported sleep duration was not validated in this study, and its stability over time is not known. Furthermore, some insomniacs may report a decrease in subjective sleep duration despite an essentially normal duration of objectively described sleep (sleep state misperception). Nonetheless, questions about self-reported sleep duration have been investigated in other studies and demonstrated to be valid measures compared to quantitative sleep assessments with actigraphy (19). Despite this, some misclassification of the sleep duration variable seems likely. However, because outcomes were assessed prospectively, any misclassification of sleep duration would likely be nondifferential with respect to diabetes. Therefore, this would tend to underestimate rather than overestimate the effects of sleep duration. Third, our study was observational in design, and thus, we cannot conclude definitively that short sleep duration caused diabetes. That is, we cannot rule out the presence of unrecognized confounders that were not accounted for in the final analysis. Fourth, because of the reduction in RRs by adding known confounders, it is possible that some residual confounding remains. However, it is doubtful that this residual unexplained confounding would be enough to completely eliminate the effect seen. Fifth, we cannot completely rule out the possibility of reverse causation. That is, short sleep duration may be a symptom of early disease, predating official diagnosis. However, analysis excluding cases diagnosed within 2 years of self-reported sleep duration did not yield appreciably different results, making this explanation unlikely. Finally, the etiology of restricted sleep was not ascertained in our study. Subjects may have had a short sleep duration from insomnia (i.e., an inability to fall or stay asleep), work/family responsibilities, or staying up late to watch television or use the internet. It is possible that the etiology of sleep restriction may differentially affect the risk of developing diabetes. This cannot be determined from our study.

CONCLUSIONS — Our data suggest that short self-reported sleep duration is associated with an increased risk of being diagnosed with diabetes. This association

persists even after adjustment for age, smoking, hypertension, and other risk factors. However, after controlling for BMI, the association was much reduced and no longer significant. This suggests that the association between a reduced self-reported sleep duration and incident diagnosis of diabetes could be due to confounding by BMI. Alternatively, given the effects of sleep deprivation on leptin metabolism, sleep restriction may mediate its effects on incident diabetes through weight gain. When analysis was confined to symptomatic cases, the results were somewhat stronger. Sleep restriction was a significant predictor of symptomatic diabetes even after controlling for BMI. This suggests that sleep restriction may predispose to the development of more severe manifestations of diabetes. Further studies are needed to better elucidate the biological mechanism underlying this association, and to determine whether the etiology of habitually short sleep (insomnia versus lifestyle choices) affects its long-term health consequences. The explanation of our finding of increased risk of diabetes in patients sleeping ≥9 h is presently unclear. Conceivably, future studies that better define these participants from a physiologic and epidemiologic standpoint could find a logical explanation for the increased risk of incident diabetes in long sleepers.

Acknowledgments— This study was supported by grants CA87969, HL24074, HL34594, RO1-HL 48531, and SCOR-P50 HL692.

We thank the participants in the Nurses Health Study for their continuing cooperation and Karen Corsano, BA, MSL, Barbara Egan, and Lisa Dunn, BS, for their expert help.

References

1. National Sleep Foundation: National Sleep Foundation sleep survey. Washington, DC, 2001
2. Terman L, Hocking A: The sleep of school children, its distribution according to age, and its relationship to physical and mental efficiency. *J Educ Psychol* 4:269–282, 1913
3. Tune G: Sleep and wakefulness in normal human adults. *Br Med J* 2:269–271, 1968
4. Bonnet M, Arand D: We are chronically sleep deprived. *Sleep* 18:908–911, 1995
5. Spiegel K, Leproult R, Van Cauter E: Impact of sleep debt on metabolic and endocrine function. *Lancet* 23:1435–1439, 1999
6. Colditz GA, Manson JE, Hankinson SE: The Nurses Health Study: 20 year contribution to the understanding of health among women. *J Women's Health* 6:49–62, 1997
7. National Diabetes Data Group: Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes* 28:1039–1057, 1979
8. Manson JE, Rimm EB, Stampfer MJ, Colditz GA, Willett WC, Krolewski A, Rosner B, Hennekens CH, Speizer FE: Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *Lancet* 338:774–778, 1991
9. Ayas NT, White DP, Manson JE, Stampfer MJ, Speizer FE, Malhotra A, Hu FB: A prospective study of sleep duration and coronary artery disease in women. Arch Intern Med. In press
10. D'Agostino RB, Lee ML, Belanger AJ, Cupples LA, Anderson K, Kannel WB: Relation of pooled logistic regression to time dependent Cox regression analysis: The Framingham Heart Study. *Stat Med* 9:1501–1515, 1990
11. Kawachi I, Colditz GA, Stampfer MJ, Willett WC, Manson JE, Speizer FE, Hennekens CH: Prospective study of shift work and risk of cornary artery disease in women. *Circulation* 92:3178–3182, 1995
12. Al-Delaimy WK, Manson JE, Willett WC, Stampfer MJ, Hu FB: Snoring as a risk factor for typeII diabetes mellitus: a prospective study. *Am J Epidemiol* 155:387–393, 2002
13. Spiegel K, Leproult R, Copinschi G, Van Cauter E: Impact of sleep length on the 24-h leptin profile (Abstract). *Sleep* 24: A74, 2001
14. Mander B, Colecchia E, Speigel K, Van Cauter E: Short sleep: a risk factor for insulin resistance and obesity (Abstract). *Sleep* 24:A74, 2001
15. Gottlieb DJ, Whitney CW, Bonekat WH, Iber C, James JD, Lebowitz M, Nieto FJ, Rosenberg CE: Relation of sleepiness to respiratory disturbance index. The Sleep Heart Health Study. *Am J Respir Crit Care Med* 159:502–507, 1999
16. Punjabi NM, Sorkin JD, Katzel CI, Goldberg AP, Schwartz AR, Smith PL: Sleep-disordered breathing and insulin resistance in middle-aged and overweight men. *Am J Respir Crit Care Med* 165:677–682, 2002
17. Kripke DF, Garfinkel L, Wingard DL, Klauber MR, Marler MR: Mortality associated with sleep duration and insomnia. *Arch Gen Psychiatr* 59:131–136, 2002
18. Expert Committee on the Diagnosis and Classification of Diabetes Mellitus: Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care* 20:1183–1197, 1997
19. Lockley SW, Skene DJ, Arendt J: Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. *J Sleep Res* 8:175–183, 1999