

## Sleep Duration as a Risk Factor for Diabetes Incidence in a Large US Sample

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**Study Objectives:** To explore the relationship between sleep duration and diabetes incidence over an 8- to 10-year follow-up period in data from the First National Health and Nutrition Examination Survey (NHANES I). We hypothesized that prolonged short sleep duration is associated with diabetes and that obesity and hypertension act as partial mediators of this relationship. The increased load on the pancreas from insulin resistance induced by chronically short sleep durations can, over time, compromise  $\beta$ -cell function and lead to type 2 diabetes. No plausible mechanism has been identified by which long sleep duration could lead to diabetes.

**Design:** Multivariate longitudinal analyses of the NHANES I using logistic regression models.

**Setting:** Probability sample (n = 8992) of the noninstitutionalized population of the United States between 1982 and 1992.

**Participants:** Subjects between the ages of 32 and 86 years.

**Measurements and Results:** Between 1982 and 1992, 4.8% of the sample (n = 430) were determined by physician diagnosis, hospital record,

or cause of death to be incident cases of diabetes. Subjects with sleep durations of 5 or fewer hours (odds ratio = 1.47, 95% confidence interval 1.03-2.09) and subjects with sleep durations of 9 or more hours (odds ratio = 1.52, 95% confidence interval 1.06-2.18) were significantly more likely to have incident diabetes over the follow-up period after controlling for covariates.

**Conclusions:** Short sleep duration could be a significant risk factor for diabetes. The association between long sleep duration and diabetes incidence is more likely to be due to some unmeasured confounder such as poor sleep quality.

**Keywords:** Sleep, diabetes, insulin resistance, obesity

**Citation:** Gangwisch JE; Heymsfield SB; Boden-Albala B; Buijs RM; Kreier F; Pickering TG; Rundle AG; Zammitt GK; Malaspina D. Sleep duration as a risk factor for diabetes incidence in a large US sample. *SLEEP* 2007;30(12):1667-1673.

ASPECTS OF OUR MODERN LIFESTYLE HAVE CONTRIBUTED TO THE INCREASING WORLDWIDE PREVALENCE OF DIABETES.<sup>1</sup> THE MOST COMMON FACTORS BELIEVED to contribute to diabetes are decreased requirements for

### Disclosure Statement

This was not an industry supported study. Dr. Heymsfield is an Executive Director, Clinical Science, at Merck & Co. This study has no relationship to his activities at Merck. Dr. Zammitt has received research support from Ancile Pharmaceuticals, Arena, Aventis, Cephalon, Elan, Epix, Forest, GlaxoSmithKline, H. Lundbeck A/S, King, Merck, Neurim, Neurocrine Biosciences, Neurogen, Organon, Orphan Medical, Pfizer, Respiroics, Sanofi-Aventis, Sanofi-Synthelabo, Schering-Plough, Sepracor, Somaxon, Takeda, UCB Pharma, Predix, Vanda, and Wyeth-Ayerst; has consulted for Aventis, Cephalon, Elan, GlaxoSmithKline, Jazz, King, Merck, Neurocrine Biosciences, Organon, Pfizer, Sanofi-Aventis, Select Comfort, Sepracor, Shire, and Takeda; has participated in speaking engagements for Neurocrine Biosciences, Kink, McNeil, SanofiAventis, Sanofi-Synthelabo, Sepracor, Takeda, Vela, and Wyeth-Ayerst; and has ownership/directorship in Clinilabs, Inc., Clinilabs IPA, Inc., and Clinilabs Physician Services, PC. The other authors have indicated no financial conflicts of interest.

Submitted for publication December, 2006

Accepted for publication August, 2007

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physical activity and ready access to highly palatable processed foods. However, there is growing evidence that another aspect of our modern lifestyle, short sleep duration, is also contributing toward the “diabetes epidemic.” The average sleep duration decreased from an estimated 9 hours in 1910<sup>2</sup> to 7 hours in 2003.<sup>3</sup> Experimental studies have shown sleep deprivation to decrease glucose tolerance and compromise insulin sensitivity.<sup>4,6</sup> Although the mechanisms by which sleep duration and diabetes risk are related are not fully understood, it has been suggested that habitually short sleep durations could lead to insulin resistance by increasing sympathetic nervous system activity,<sup>7,8</sup> raising evening cortisol levels,<sup>9</sup> and decreasing cerebral glucose utilization.<sup>5,10</sup> The increased burden on the pancreas from insulin resistance can, over time, compromise  $\beta$ -cell function and lead to type 2 diabetes.

Previous epidemiologic studies on the relationship between sleep duration and diabetes have shown varying results. Table 1 shows a summary of the 4 longitudinal and 2 cross-sectional currently published studies that examined this relationship. Ayas et al<sup>11</sup> found both long ( $\geq 9$  hours) and short ( $\leq 5$  hours) sleep durations to be significantly associated with diabetes incidence in multivariate models adjusting for a variety of potentially confounding variables, but, after adding body mass index (BMI) to the models, only long sleep durations were significantly associated with diabetes incidence. When the investigators performed similar analyses with only symptomatic cases of diabetes, they found both long and short sleep durations to be significantly associated with diabetes incidence. The study by Bjorkelund et al<sup>12</sup>

**Table 1**—Summary of Epidemiologic Studies that Examined the Association Between Sleep Duration and Diabetes Incidence

<b>Longitudinal</b>			
<b>Source</b>	<b>Sample</b>	<b>Follow-up period, y</b>	<b>Covariates in multivariate analyses</b>
Ayas et al <sup>11</sup>	70,026 U.S. women aged 40-65 y	10	Physical activity, depression, alcohol, age, smoking, BMI, hypertension, shiftworking, hypercholesterolemia, snoring, postmenopausal hormone use, family history of diabetes
Bjorkelund et al <sup>12</sup>	661 Swedish women aged 38-60 y	32	Age, BMI, waist-hip ratio, subscapular skinfold
Mallon et al <sup>13</sup>	1187 Swedish women and men aged 45-65 y	12	Depression, alcohol, age, obesity, hypertension, smoking, not married, living alone, snoring, sex
Yaggi et al <sup>14</sup>	1139 US men aged 40-70 y	15-17	Education, age, hypertension, smoking, self-rated health status, waist circumference, testosterone
<b>Cross-Sectional</b>			
<b>Source</b>	<b>Sample</b>		<b>Covariates in multivariate analyses</b>
Gottlieb et al <sup>15</sup>	1486 US diabetic and nondiabetic women and men, aged 53-93 y		Age, sex, ethnicity, waist girth, apnea-hypopnea index
Knutson et al <sup>16</sup>	161 US diabetic African-American women and men; average age = 57 ± 12 y		Age, sex, BMI, insulin use, presence of major complications

BMI refers to body mass index.

found no relationship between sleep duration and the incidence of diabetes. Mallon et al<sup>13</sup> found sleep durations of 5 or fewer hours to be associated with diabetes incidence only in men, and sleep durations of 9 or more hours to be associated with diabetes incidence only in women, although the association in women was not statistically significant, possibly due to a lack of statistical power from having few female subjects who reported sleeping for long durations. Both the longitudinal study by Yaggi et al<sup>14</sup> and the cross-sectional study by Gottlieb et al<sup>15</sup> showed U-shaped relationships between sleep duration and diabetes, with both short and long sleep durations being significantly associated with diabetes in multivariate models. In a cross-sectional study with subjects who had type 2 diabetes, Knutson et al<sup>16</sup> found a significant association between higher perceived sleep debt and poorer glycemic control.

The aim of the current study is to explore the relationship between sleep duration and the diagnosis of diabetes over an 8- to 10-year follow-up period between 1982 and 1992 among subjects who participated in the Epidemiologic Follow-up Studies of the first National Health and Nutrition Examination Survey (NHANES I).<sup>17-20</sup> We hypothesized that prolonged short sleep duration is associated with diabetes. We theorized that obesity and hypertension, potent risk factors for diabetes, act as partial mediators of this relationship, since short sleep duration is associated with obesity<sup>21,22</sup> and with the incidence of hypertension.<sup>23</sup>

## METHODS

### Study Population

Subjects for this study were participants in the 1982-1984, 1986, 1987, and 1992 Epidemiologic Follow-up Studies of the NHANES I. The NHANES I Epidemiologic Follow-up Study is a longitudinal study of adults originally examined and interviewed in 1971 to 1975 as part of the NHANES I. The primary purpose of the follow-up study is to investigate longitudinal relationships between physi-

ologic, nutritional, and behavioral characteristics collected during NHANES I and subsequent morbidity and mortality from specific diseases and conditions. The 1982 to 1984 wave of data collection followed all medically examined respondents who had been 25 to 74 years of age in 1971 to 1975 (n = 14,407). Eighty-five percent of all eligible subjects were successfully recontacted (n = 12,220). The measures of self-reported sleep duration were taken from the 1982-1984 survey when the following question was asked: "How many hours of sleep do you usually get a night (or when you usually sleep)?" Individuals who were deceased (n = 1697), who did not answer the sleep duration question (n = 734), and who had diabetes at or before the 1982 to 1984 survey (n = 797) were excluded from the analyses, yielding a final sample size of 8992 subjects. The 1986 wave focused on subjects who had been 55 to 74 years of age at their baseline examinations in 1971 to 1975, whereas the 1987 and 1992 waves followed all medically examined respondents who had been 25 to 74 years of age in 1971 to 1975. Diabetes incidence was determined over an 8- to 10-year follow-up period from interviews conducted with all subjects traced and interviewed at the 1986, 1987, and 1992 Follow-up Studies.

To see whether the subjects (n = 8992) included in our study in 1982 to 1984 differed substantially from the NHANES I cohort (n = 14,407) in 1971 to 1975, we performed analyses on baseline variables to determine their effects upon their ultimate inclusion in the sample in 1982 to 1984. We categorized age at baseline in 1971 to 1975 by 5-year age increments and found that rates of loss to follow-up were highest among participants aged 60 years and older. Rates of loss to follow-up were also higher among men (43% for men vs 34% for women), nonwhites (51% for nonwhites vs 35% for whites), those who had not graduated from high school (48% for < high school graduate vs 29% for ≥ high school graduate), those who were unmarried (34% for married vs 48% for unmarried), and those who were underweight, overweight, and obese (46% for underweight, 33% for normal weight, 39% for overweight, 46% for obese, and 51% for morbidly obese).

**Table 2**—Baseline Characteristics and Risk Factors for Diabetes by Self Reported Sleep Duration

Baseline characteristics and risk factors for diabetes	Hours of Sleep					F (P value)
	≤ 5	6	7	8	≥ 9	
n (%)	802 (8.9)	1799 (20.0)	2674 (29.7)	2936 (32.6)	781 (8.7)	
Age, y	58.4 (14.7)	54.9 (14.2)	53.6 (13.5)	56.4 (14.7)	63.4 (15.8)	80.3 (<0.0001)
BMI, kg/m <sup>2</sup>	27.0 (7.5)	26.7 (7.8)	26.0 (5.9)	26.1 (6.5)	26.6 (10.5)	5.9 (0.0001)
PA	3.7 (1.2)	4.0 (1.1)	4.0 (1.1)	4.0 (1.1)	3.7 (1.1)	31.1 (<0.0001)
						<b>X<sup>2</sup> (P value)</b>
Depression	33.8	19.3	12.7	15.3	20.5	209.2 (<0.0001)
AA	44.5	36.0	34.1	42.0	53.5	122.5 (<0.0001)
Hypertension	66.6	57.6	51.7	55.5	65.2	84.9 (<0.0001)
Women	65.1	61.5	62.9	63.7	61.7	4.5 (0.3489)
Nonwhite	20.2	16.2	10.4	12.3	16.5	73.6 (<0.0001)
≥ HS Grad	51.9	65.4	71.1	62.6	46.9	209.8 (<0.0001)

Data are presented as mean (SD) for age, body mass index (BMI), and physical activity (PA) and as percentages for depression, alcohol abstinence (AA), hypertension, being a woman, being nonwhite, and having an education level of high school graduate (HS grad) or higher.

This study involved analyses of a publicly available dataset that did not include identifying information and, therefore, met federal guidelines for exemption from Institutional Review Board review.

### Assessment of Diabetes

Individuals who self-reported a physician diagnosis of diabetes at or before the 1982-1984 survey were excluded from the analyses. Incident cases of diabetes over the 8- to 10-year follow-up period were determined by self-report of physician diagnosis, by hospital diagnosis, or by cause of death, at the times of the 1986, 1987, or 1992 follow-up surveys. The validity of self-reported physician-diagnosed diabetes has been shown previously.<sup>24</sup> Any hospital-stay record with an ICD-9 code under category 250 for diabetes mellitus was included, whether the disease was considered primary or secondary.

### Assessment of Covariates

The 1982-1984 questionnaire included questions on body weight (BMI = kg/m<sup>2</sup>: lean < 25, overweight ≥ 25 and < 30, and obese ≥ 30), history of hypertension (yes, no), physical activity (6: high; 5, 4, 3, 2: low), depression (yes, no), alcohol consumption (0, > 0 and < 28, ≥ 28 g/day), ethnicity (white or nonwhite, including black, Hispanic, Asian, and other), education (high school graduate or < high school graduate), marital status (married, unmarried), and age (5-year interval). To measure the presence of depressive symptoms, we used the standard cutoff score of 16 out of a total possible score of 60 on the 20-question Center for Epidemiologic Studies Depression Scale.<sup>25</sup> Sex was not found to be associated in bivariate analysis with either the outcome of diabetes incidence or with the exposure of sleep duration and was therefore not included as a covariate in the multivariate analyses. Depression was not found in bivariate analyses to be significantly associated with diabetes incidence in this dataset. We chose to include depression as a covariate in the multivariate analyses because it is strongly associated with sleep duration, because there are strong theoretical mechanistic links between depression and diabetes,<sup>26</sup> and because depression has been found to be associated with diabetes in other studies.<sup>27</sup> The subjects' levels of physical activity were measured by adding the scores from 2 questions that

asked them to estimate how much physical activity they obtained in recreational and nonrecreational activities. They were given a score of 3 if they were very active, 2 if they were moderately active, and 1 if they were inactive. Scores therefore ranged from 2 to 6, with increasing scores representing increased levels of physical activity. To compute BMI (kg/m<sup>2</sup>), we used heights from medical examinations conducted between 1971 and 1975 and actual body weights measured with scales at the 1982 to 1984 interviews. For subjects with missing values on measured body weight, we substituted their self-reported body weight for measured body weight. The measured and self-reported body weights obtained in 1982 to 1984 for the entire sample had a Pearson correlation coefficient of 0.975, indicating a reasonable level of accuracy for the self-reported weights. Missing values for covariates, which for all covariates represented less than 1% of the total sample size, were imputed using mean and mode substitution.

### Statistical Analyses

After performing preliminary univariate and bivariate analyses, we used logistic regression models to examine the effect of sleep duration upon the risk for incident diabetes over the 8- to 10-year follow-up period. We chose 7 hours as the reference category for 3 reasons. First, 7 hours of sleep has consistently been shown to have the lowest mortality risk.<sup>28-30</sup> Second, the mean and median sleep durations are 7 hours in this sample and in adults in the US.<sup>3</sup> Third, the choice of this category eased interpretation of the odds ratios (OR), since subjects who reported getting 7 hours of sleep had the lowest incidence of diabetes. Covariates in the first adjusted multivariate model (Model 2) included physical activity, depression, alcohol consumption, ethnicity, education, marital status, and age. We included BMI and history of hypertension in the final model (Model 3) to test whether these variables acted as partial mediators of the relationship between sleep duration and the incidence of diabetes. The significance of individual coefficients in the logistic regression models were determined by the 95% confidence limits for OR.

The NHANES I included weights to account for the complex sampling design and to allow approximations of the US population. We conducted both unweighted analyses using SAS Software<sup>31</sup> and weighted analyses using SUDAAN<sup>32</sup> Software. We chose to present only the unweighted results for 4 reasons. First,

the weighted results were generally consistent with minimal effects of the complex survey design on the main conclusions derived from the unweighted estimates. Second, our objective was not to provide national estimates, but to look at the relationship between sleep duration and the incidence of diabetes. Third, our study's baseline measures were taken from the 1982 to 1984 Follow-up to the NHANES I, so the weights created for baseline measures taken from the 1971 to 1975 NHANES I did not account for subjects who were lost to follow-up between the 2 waves. Fourth, there have been differences of opinion regarding the appropriateness of using the sample weights with the NHANES.<sup>33</sup>

## RESULTS

Table 2 shows the baseline characteristics of the entire sample according to the sleep-duration categories. Sleep durations of 5 hours or less were associated with older age, higher BMI, lower physical activity, depression, abstinence from alcohol, diagnosis of hypertension, non-white ethnicity, and less than a high school graduate education. Sleep durations of 9 or more hours were associated with older age, higher BMI, lower physical activity, depression, abstinence from alcohol, diagnosis of hypertension, non-white ethnicity, and less than a high school graduate education.

Table 3 shows the OR of incident diabetes over the 8- to 10-year follow-up period, as computed with logistic regression models. There were 430 incident cases of diabetes over this period. In the unadjusted results (Model 1), subjects who reported sleeping 5 or fewer hours (OR = 1.91, 95% CI 1.37-2.67) and subjects who reported sleeping 9 or more hours (OR = 1.85, 95% CI 1.32-2.60) were significantly more likely to have incident diabetes over the follow-up period than were subjects who reported sleeping 7 hours. After adjusting for physical activity, depression, alcohol consumption, ethnicity, education, marital status, and age (Model 2), those who slept 5 or fewer hours (OR = 1.57, 95% CI 1.11-2.22) and those who slept 9 or more hours (OR = 1.57, 95% CI 1.10-2.24) continued to be significantly more likely to have incident diabetes. Consistent with our hypothesis that BMI and history of hypertension would act as partial mediators of the relationship between sleep duration and the incidence of diabetes, the addition of these variables in Model 3 further attenuated the results. Subjects who reported getting 5 or fewer hours of sleep (OR = 1.47, 95% CI 1.03-2.11) and those who reported getting 9 or more hours (OR = 1.52, 95% CI 1.06-2.17) continued to be significantly more likely to have incident diabetes after controlling for obesity, history of hypertension, and the other covariates.

## DISCUSSION

We found a U-shaped relationship between sleep duration and diabetes incidence, with both short and long sleep durations being associated with diabetes incidence. The physiologic mechanisms likely to explain the associations between short sleep duration and diabetes incidence and long sleep duration and diabetes incidence are quite distinct.

Subjects who slept 5 or fewer hours were almost twice as likely as those who slept 7 hours to have incident diabetes over the follow-up period. Controlling for the potential confounding variables attenuated this relationship. The relationship was further attenuated with the inclusion of body weight and hypertension in the multivariate models. The effect of short sleep duration on dia-

**Table 3**—Odds ratios (95% confidence interval) of diabetes incidence over the follow-up period by sleep duration at baseline

Hours of Sleep	Model 1 <sup>a</sup>	Model 2 <sup>b</sup>	Model 3 <sup>c</sup>
≤ 5	1.91 (1.37-2.67)	1.57 (1.11-2.22)	1.47 (1.03-2.09)
6	1.25 (0.94-1.70)	1.19 (0.88-1.60)	1.08 (0.80-1.47)
7	1.00	1.00	1.00
8	1.16 (0.89-1.51)	1.09 (0.84-1.43)	1.09 (0.83-1.43)
≥ 9	1.85 (1.32-2.60)	1.57 (1.10-2.24)	1.52 (1.06-2.17)

<sup>a</sup>Model 1 – Unadjusted.

<sup>b</sup>Model 2 – Adjusted for physical activity, depression, alcohol consumption, ethnicity, education, marital status, and age.

<sup>c</sup>Model 3 – Adjusted for the variables in Model 2 plus overweight/obesity and hypertension.

betes incidence is therefore likely to be related in part to the influence of short sleep duration upon body weight and hypertension. Chronic sleep deprivation is theorized to lead to weight gain and obesity by compromising insulin sensitivity and by increasing appetite by decreasing leptin levels and increasing ghrelin levels.<sup>10,34</sup> Prolonged exposure to short sleep durations is hypothesized to lead to hypertension by raising average 24-hour blood pressure and heart rate and by elevating sympathetic nervous system activity, which could lead to structural adaptations that gradually reset the entire cardiovascular system to operate at an elevated pressure equilibrium.<sup>23</sup>

The association between short sleep duration and diabetes incidence continued to be statistically significant after controlling for body weight and hypertension. Short sleep duration is therefore likely to have direct effects upon the risk for the incidence of diabetes independent of its influence upon body weight and blood pressure. These results are consistent with findings from studies showing sleep deprivation to decrease glucose tolerance and compromise insulin sensitivity.<sup>4,6</sup> Sleep deprivation has been theorized to adversely impact glucose tolerance through a number of pathways.<sup>10</sup> First, sleep deprivation has been shown in humans to increase sympathetic nervous system activity, as evidenced by increased urinary and plasma catecholamine levels.<sup>7,8</sup> Second, sleep deprivation has been shown in positron emission tomography scans to decrease cerebral glucose utilization.<sup>5</sup> The brain is a major source of non-insulin-dependent glucose utilization, so decreased brain glucose uptake may result in higher circulating glucose concentrations, which over time could facilitate the development of insulin resistance.<sup>10</sup> Third, acute sleep deprivation has been shown to raise evening levels of cortisol, an insulin antagonist, that, under conditions of chronic sleep loss could, compromise insulin sensitivity in peripheral sites.<sup>9</sup> Insulin resistance and the resultant increased load on the pancreas can lead to type 2 diabetes over time by compromising  $\beta$ -cell function.

The OR for the association between short sleep duration and incident diabetes were under 2.0 in all of the logistic models. This relatively modest association could be due to a number of factors. First, the influence of short sleep duration on the development of diabetes can be mitigated or exacerbated by the presence or

absence of other risk and protective factors for the disease, such as exercise and diet. Physical exercise has been shown to lessen the effects of sleep deprivation on insulin sensitivity.<sup>4</sup> Sleep restriction has been shown in laboratory studies to increase appetite, with particular cravings for sweet and starchy snacks,<sup>34</sup> but increased appetite does not necessarily result in increased consumption. If consumption does increase, then characteristics of the foods consumed, such as glycemic index, fiber content, and caloric content, can affect the ultimate influence upon weight gain and insulin sensitivity. Nutritional consumption has not been controlled for in any of the epidemiologic studies on the relationship between sleep duration and diabetes. Second, genetically determined characteristics or traits could result in differential vulnerability to the effects of short sleep duration upon insulin sensitivity and appetite. Some individuals might therefore be at increased risk for the development of diabetes in response to chronically short sleep durations. Evidence has been found for the presence of trait-like differential vulnerability to neurobehavioral impairment from sleep deprivation.<sup>35</sup>

The association between short sleep duration and diabetes is consistent with physiologic data from experimental sleep-restriction studies, but elucidating the mechanisms mediating the association between long sleep duration and diabetes has posed a conundrum. Investigators from the Nurses' Health Study stated that "excessive sleep per se could directly lead to an increased risk of diabetes," but the authors "knew of no plausible physiologic explanation for such a cause-and-effect relationship."<sup>11</sup> Researchers from the Massachusetts Male Aging Study stated that "physiological evidence to explain the increased risk associated with > 8 hours of sleep is absent, but it could be due to confounding by some unmeasured variable."<sup>14</sup>

One plausible explanation for the association between long sleep duration and diabetes is the sleep inducing and metabolic effects of proinflammatory cytokines.<sup>15</sup> Proinflammatory cytokines have been shown to have deleterious effects on both glucose homeostasis and  $\beta$ -cell function<sup>36</sup> and have been found to be elevated in obesity and in conditions in which the primary pathogenic mechanism is insulin resistance.<sup>37</sup> Proinflammatory cytokines contribute toward sleepiness and fatigue and are believed to play key roles in sleep pathology, such as sleep apnea and sleepiness.<sup>38</sup> The sleep-inducing effects of proinflammatory cytokines are presumed to be an evolutionary adaptation to promote rest and recovery from illness. According to this view, long sleep duration is not considered to be a cause of diabetes but, rather, a consequence of diabetes and other conditions associated with chronic inflammation.

Poor sleep quality from sleep disorders could also have played a part in the association between long sleep duration and diabetes incidence.<sup>11,14</sup> Sleep-disordered breathing, characterized by frequent microarousals and reductions in slow-wave sleep, is associated with diabetes.<sup>39</sup> The NHANES I Follow-up Survey did not include questions on sleep disorders, but we would expect that individuals with sleep-disordered breathing would be more likely to self-report higher average sleep times, since they are often unaware of their disrupted sleep patterns and require longer sleep durations to compensate for poor sleep quality. Experimental induction of microarousals and suppression of slow-wave sleep without changes in total sleep time have been found to be associated with decreased insulin sensitivity and increased daytime sympathetic activity.<sup>40</sup> Increased time in bed to compensate for

poor sleep quality could therefore have been a factor in the association between long sleep duration and diabetes incidence.

The relationship between sleep duration and diabetes incidence could be reflective of disturbances in the suprachiasmatic nucleus (SCN). The SCN generates and organizes autonomic rhythms that coordinate the transition of the body from inactive phases to active phases through modulation of the parasympathetic and sympathetic nervous systems. The SCN evolved to rely upon repeated physiologic cues from both the external and the internal environments to synchronize rest, activity, and consumption to the circadian and seasonal cycles that change in a precise and predictable fashion. In industrialized countries, dramatic alterations in the timing and duration of sleep, activity, and feeding during the last century could disturb the functioning of the SCN in susceptible individuals. The SCN has been shown to connect directly via the autonomic nervous system to each of the metabolic organs that malfunction in diabetes: pancreas, liver, and adipose tissue.<sup>41-43</sup> A disturbed SCN could contribute toward diabetes through the paradoxical concurrent dominance of the parasympathetic and sympathetic systems in different metabolic organs.<sup>44</sup>

Short sleep duration could also influence diabetes incidence by making it more difficult to engage in behaviors that could be protective against the disease. Inadequate sleep has been shown to be associated with pessimism, impatience, irritability, and feeling tired and stressed.<sup>45</sup> These feelings and emotional states could reduce one's resolve and willpower to follow guidelines for physical activity and nutrition that could help prevent the onset of diabetes. We found that subjects with both short and long sleep durations had lower levels of physical activity.

When interpreting the results from this study, we must keep in mind that the baseline measure of sleep duration was obtained over 20 years ago. The average sleep duration in this cohort was 7.13 (SD = 1.29) hours; whereas respondents to the 2005 National Sleep Foundation's "Sleep in America Poll" reported sleeping an average of 6.8 hours on weekdays and 7.4 hours on weekends.<sup>46</sup> Average sleep durations are likely to have decreased since the early 1980s due to societal and technologic changes, including increases in shift work, cable television, use of the internet, 24-hour stores, and dual-income families.

Although the findings from this study show a relationship between sleep duration and diabetes incidence, a number of limitations to these analyses must be considered as well. Important questions are whether reverse causation contributed toward these results or whether some uncontrolled confounder played a part in the findings. We cannot rule out the possibility that short or long sleep duration may be a prodromal symptom of diabetes that predates diagnosis. Another limitation of this study was the use of self-reported sleep durations as opposed to measured sleep durations. Good agreement has been found though in previous studies between self-reported sleep durations and those obtained through actigraphic monitoring.<sup>47,48</sup> The NHANES I also lacked repeated measures of sleep duration, so we were unable to determine how representative the baseline sleep measure was of the sleep durations over the follow-up period. Changes in sleeping patterns over the follow-up period could have weakened the association between sleep duration reported at baseline and subsequent diabetes incidence. Diabetes also frequently goes undiagnosed, and we have no way of knowing whether the subjects' likelihood of seeking or receiving treatment, and therefore being diagnosed with diabetes, varied differentially by sleep duration. Other limi-

tations include possible bias arising from loss to follow-up and missing data on baseline risk variables.

The results from this study suggest that short sleep duration could play a role in the etiology of diabetes in some individuals. If short sleep duration functions to increase insulin resistance and decrease glucose tolerance, then interventions that increase the amount and improve the quality of sleep could potentially serve as treatments and as primary preventative measures for diabetes. Examples of behavioral interventions include helping patients to modify maladaptive sleep habits and educating them about healthier sleep hygiene practices. Further research is needed to investigate the biologic mechanisms that link short sleep duration and diabetes and to explore the efficacy of sleep interventions for the treatment and prevention of diabetes. We are not aware of any plausible physiologic explanations whereby long sleep duration could play a role in the pathogenesis of diabetes. It is more likely that long sleep duration occurs in parallel to, and as a consequence of, diabetes and other conditions associated with chronic inflammation. Future studies on the relationship between sleep duration and diabetes should include assessments of sleep quality to elucidate whether long sleep duration is associated with poor sleep quality and measures of inflammatory cytokines to see whether long sleep duration is associated with these regulatory proteins.

## ACKNOWLEDGMENTS

Financial support for this study was provided by National Research Service Award T32 MH013043 from the NIH/National Institute of Mental Health to Columbia University's Psychiatric Epidemiology Research Training Program and by R24 HL76857 from the NIH/National Heart Blood and Lung Institute to Columbia University's Behavioral Cardiovascular Health & Hypertension Program.

Institution at which work was performed: Columbia University

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