Association of Sleep Time With Diabetes Mellitus and Impaired Glucose Tolerance

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Background: Experimental sleep restriction causes impaired glucose tolerance (IGT); however, little is known about the metabolic effects of habitual sleep restriction. We assessed the cross-sectional relation of usual sleep time to diabetes mellitus (DM) and IGT among participants in the Sleep Heart Health Study, a community-based prospective study of the cardiovascular consequences of sleep-disordered breathing.

Methods: Participants were 722 men and 764 women, aged 53 to 93 years. Usual sleep time was obtained by standardized questionnaire. Diabetes mellitus was defined as a serum glucose level of 126 mg/dL or more (≥7.0 mmol/L) fasting or 200 mg/dL or more (≥11.1 mmol/L) 2 hours following standard oral glucose challenge or medication use for DM. Impaired glucose tolerance was defined as a 2-hour postchallenge glucose level of 140 mg/dL or more (≥7.8 mmol/L) and less than 200 mg/dL. The relation of sleep time to DM and IGT was examined using categorical logistic regression with adjustment for age, sex, race, body habitus, and apnea-hypopnea index.

Results: The median sleep time was 7 hours per night, with 27.1% of subjects sleeping 6 hours or less per night. Compared with those sleeping 7 to 8 hours per night, subjects sleeping 5 hours or less and 6 hours per night had adjusted odds ratios for DM of 2.51 (95% confidence interval, 1.57-4.02) and 1.66 (95% confidence interval, 1.15-2.39), respectively. Adjusted odds ratios for IGT were 1.33 (95% confidence interval, 0.83-2.15) and 1.58 (95% confidence interval, 1.15-2.18), respectively. Subjects sleeping 9 hours or more per night also had increased odds ratios for DM and IGT. These associations persisted when subjects with insomnia symptoms were excluded.

Conclusions: A sleep duration of 6 hours or less or 9 hours or more is associated with increased prevalence of DM and IGT. Because this effect was present in subjects without insomnia, voluntary sleep restriction may contribute to the large public health burden of DM.

Arch Intern Med. 2005;165:863-868

THE USUAL AMOUNT OF SLEEP per night has been declining among US adults for more than a generation. The median sleep time in adults aged 40 to 79 years was 8 hours per night in 1959, with less than 15% reporting a usual sleep time of less than 7 hours.[1] By 2002, the adult median sleep time had decreased to 7 hours per night, with more than one third of adults sleeping fewer than 7 hours.[2] Although insomnia is highly prevalent, much of the reduction in sleep time reflects voluntary sleep restriction, with 43% of adults reporting that they often stay up later than they should watching television or using the Internet and 45% reporting that they sleep less to get more work done.[3] Several studies have found increased mortality associated with usual sleep times of less than 7 or more than 8 hours per night.[4-7] Experimental restriction of sleep to 4 hours per night for 6 nights resulted in impaired glucose tolerance (IGT) in healthy young adults.[8] Because diabetes mellitus (DM) carries a high risk of cardiovascular-related mortality, the impact of sleep restriction on glucose regulation suggests a mechanism whereby short sleep time might increase mortality. In the present study, we examined the relation of self-reported usual sleep time to prevalent DM and IGT in a large community-based sample of middle-aged and older adults.

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Financial Disclosure: None.

METHODS

STUDY SAMPLE

Subjects were participants in the Sleep Heart Health Study (SHHS), a community-based, prospective, cohort study of the cardiovascular consequences of obstructive sleep apnea/hypopnea,[9] who were recruited independent of the presence or absence of obstructive sleep

apnea/hypopnea. The 6441 SHHS participants completed a Sleep Habits Questionnaire and underwent overnight polysomnography between 1995 and 1998. The Atherosclerosis Risk in Communities Study and the Cardiovascular Health Study, 2 parent cohorts from which subjects were recruited into the SHHS, had performed oral glucose tolerance tests on their participants in proximity to the baseline SHHS examination. Subjects in the present analysis are SHHS participants from the Atherosclerosis Risk in Communities Study and the Cardiovascular Health Study cohorts whose glycemic status was assessed within 12 months following the baseline SHHS examination. Subjects were excluded if they had missing data for age, sex, race, body mass index (BMI) (calculated as weight in kilograms divided by the square of height in meters), waist girth, apnea-hypopnea index (AHI), or sleep time. The protocol was approved by the institutional review board of each participating center, and signed informed consent was provided by each subject.

SLEEP HABITS QUESTIONNAIRE

Usual sleep time was defined as the response to the question, "How many hours of sleep do you usually get at night (or your main sleep period) on weekdays or workdays?" Responses were integer values. We assessed the stability of self-reported sleep time in 2117 subjects completing a second Sleep Habits Questionnaire after a mean (SD) interval of 2.4 (0.5) years. The median reported sleep time was unchanged at 7 hours, and the median absolute difference in sleep time was 1 hour (interquartile range, 0-1 hour). Sleep times reported at baseline and follow-up were significantly, although modestly, correlated (r_s=0.57, $P<.001$), with the absolute difference in reported sleep time increasing slightly, but significantly ($P<.001$), with the length of time between successive administrations of the questionnaire.

Symptoms of insomnia were obtained from responses on a 5-point Likert scale to the items "Have trouble falling asleep," "Wake up during the night and have difficulty getting back to sleep," "Wake up too early in the morning and be unable to get back to sleep," and "Take sleeping pills or other medication to help you sleep." Response options were as follows: never, rarely (≤1 time per month), sometimes (2-4 times per month), often (5-15 times per month), and almost always (16-30 times per month). For analysis, these variables were collapsed into 2 categories: infrequent, comprising the responses never, rarely, and sometimes; and frequent, comprising the responses often and almost always. Insomnia was operationally defined as a "frequent" response to any of these 4 questions. Frequencies of nocturia, nocturnal leg jerks or cramps, and nocturnal joint pain were similarly obtained.

DM AND GLUCOSE INTOLERANCE

Use of insulin or oral hypoglycemic agents to treat DM was ascertained by each parent study. Morning blood samples were obtained by venipuncture after an overnight fast of at least 8 hours, and extracted plasma was stored at −70°C for later glucose determination by the hexokinase method. Following venipuncture, subjects were given a 75-g oral glucose load, and venipuncture was repeated after 2 hours to obtain a postload glucose value. Diabetes mellitus and IGT were defined in accordance with American Diabetes Association and World Health Organization guidelines.[10,11] Diabetes mellitus was defined as use of insulin or a hypoglycemic agent, a fasting plasma glucose level of 126 mg/dL or more (≥7.0 mmol/L), or a 2-hour postload plasma glucose level of 200 mg/dL or more (≥11.1 mmol/L). Impaired glucose tolerance was defined as a 2-hour postload plasma glucose level of 140 mg/dL or more (≥7.8 mmol/L) in subjects not meeting the criteria for DM.

OTHER MEASURES

Body mass index and waist girth measured at the level of the umbilicus were ascertained by each parent cohort at the clinic examination at which DM and IGT were assessed. Usual daily alcohol consumption was also ascertained by each parent cohort. Unattended in-home polysomnography was performed by the SHHS, and records were centrally scored.[12] The AHI was defined as the number of apneas plus hypopneas, each associated with at least a 4% decrease in oxyhemoglobin saturation, per hour of sleep. A standardized health interview administered before polysomnography was used to obtain data on smoking habits, daily caffeine consumption, physician-diagnosed angina, myocardial infarction, stroke, heart failure, and history of coronary revascularization procedures. Symptoms of depression were obtained from 2 questions on the Medical Outcomes Study 36-Item Short-Form Health Survey[13]: "During the past 4 weeks, how much of the time . . . " (1) "Have you felt so down in the dumps that nothing could cheer you up?" and (2) "Have you felt downhearted and blue?" Responses from the 6-point Likert scale were collapsed into 2 categories: "none," "a little," or "some" of the time vs "all," "most," or "a good bit" of the time. Medication use and blood pressure were ascertained using previously described procedures.[14,15]

STATISTICAL ANALYSIS

Unadjusted differences in continuous and categorical variables across sleep time categories were assessed for significance using single-factor analysis of variance or contingency table analysis, as appropriate. General categorical logistic regression analysis was implemented using a procedure (PROC CATMOD) in SAS statistical software, version 8.1 (SAS Institute Inc, Cary, NC), to assess the relation of usual sleep time to DM and IGT, adjusting for relevant covariates. Covariates included in the models were age, age^2, waist girth, and AHI as continuous measures, along with sex, race (non-Hispanic white vs all other), and parent cohort as categorical variables. Because of the positively skewed distribution of the AHI, it was log transformed as ln(AHI + 1). Additional terms, including BMI and quadratic terms for waist, BMI, and ln(AHI + 1), were also considered in the models, but were not included because they did not significantly improve the model fit and had no meaningful influence on the outcome of interest. Secondary analyses stratified by sex, age, and AHI were performed to assess the consistency of results across the study sample. For these analyses, an outcome combining DM and IGT was used to obtain stable effect estimates within strata. To assess the contribution of insomnia to the observed effects, secondary analyses adjusting for insomnia symptoms or stratified by the presence or absence of insomnia symptoms were also performed (n = 1477 subjects with complete data).

RESULTS

Of 3168 SHHS participants from the Atherosclerosis Risk in Communities Study and the Cardiovascular Health Study parent cohorts, 1682 were excluded for missing data (47 had a missing sleep time, 1622 had missing glucose tolerance data, and 13 had missing body habitus data). Among the 1486 included subjects (722 men and 764 women), the mean age was 70.2 years (SD, 8.5 years; range, 53-93 years), the mean BMI was 28.1 (SD, 4.7), and the mean AHI was 9.7 (SD, 12.5; median, 5.4). Compared with the included subjects, excluded subjects had a slightly lower mean (SD) age (67.9 [9.3] years) and AHI (8.8 [12.5]), and were more

Table 1. Characteristics of the 1486 Study Participants

Characteristic	Reported Usual Sleep Time per Night, h*				*P* Value†
	≤5	6	7-8	≥9	
No. of subjects	125	277	956	128	NA
Age, y‡	71.3 (8.1)	70.4 (8.5)	69.7 (8.6)	72.4 (8.0)	.002
Female sex	58.4	54.5	49.5	52.3	.17
Minority race	7.2	7.9	6.2	8.6	.60
BMI‡	28.2 (5.2)	28.2 (5.0)	28.1 (4.6)	27.7 (4.6)	.76
Waist girth, cm‡	100.3 (13.8)	100.3 (13.5)	100.6 (12.2)	99.5 (13.4)	.84
AHI, events/h§	5.6 (2.0-12.5)	5.0 (1.7-12.4)	5.4 (1.9-12.2)	5.4 (1.5-12.8)	.85
Glucose regulation‖					
Normal glucose tolerance	41.6	45.1	55.3	39.8	<.001
Impaired glucose tolerance	25.6	31.8	26.5	35.9	
Diabetes mellitus	32.8	23.1	18.2	24.2	
Often or always . . . ¶					
Have trouble falling asleep	51.6	21.2	8.8	7.0	<.001
Wake up too early in the morning	62.1	30.7	10.3	6.3	<.001
Wake up and cannot return to sleep	65.3	29.9	13.0	9.4	<.001
Take a pill to help sleep	11.3	9.9	5.9	10.2	.02
Alcohol consumption ≥2 drinks/d	4.8	4.7	7.4	12.5	.03

Abbreviations: AHI, apnea-hypopnea index; BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); NA, data not applicable.

*Data are given as percentage of subjects in each group (for the categorical characteristics) unless otherwise indicated.

†Significance tests for the unadjusted difference across categories of sleep time are based on the χ^2 test for contingency table analysis of categorical characteristics and on analysis of variance for continuous characteristics, using ln(AHI + 1) to test for differences in AHI across categories.

‡Data are given as mean (SD) (for the continuous characteristics).

§Data are given as median (interquartile range) (for another continuous characteristic).

‖Percentages may not total 100 because of rounding.

¶Because of missing data, the number of subjects with data for this characteristic is 1477 (124, 274, 951, and 128 for sleep time categories of ≤5, 6, 7-8, and ≥9 hours per night, respectively).

Table 2. Data for Diabetes Mellitus and Impaired Glucose Tolerance by Reported Usual Sleep Time in 1486 Subjects

Usual Sleep Time per Night, h	Model*					
	1		2		3	
	DM	IGT	DM	IGT	DM	IGT
≤5	2.40 (1.54-3.74)	1.29 (0.81-2.05)	2.45 (1.55-3.88)	1.31 (0.82-2.11)	2.51 (1.57-4.02)	1.33 (0.83-2.15)
6	1.56 (1.10-2.20)	1.47 (1.08-2.01)	1.64 (1.15-2.35)	1.57 (1.14-2.15)	1.66 (1.15-2.39)	1.58 (1.15-2.18)
≥9	1.85 (1.15-2.98)	1.89 (1.23-2.89)	1.75 (1.07-2.85)	1.84 (1.19-2.84)	1.79 (1.08-2.96)	1.88 (1.21-2.91)

Abbreviations: DM, diabetes mellitus; IGT, impaired glucose tolerance.

*Data are given as odds ratios (95% confidence intervals) for the presence of DM or IGT relative to normal glucose tolerance, from categorical logistic regression models using 7 to 8 hours of sleep per night as the referent category. $P<.001$ for all 3 models, reflecting the overall significance level of the effect of sleep time on DM and IGT, based on the likelihood ratio χ^2_6. Model 1 was unadjusted; 2, adjusted for age, age^2, sex, race/ethnicity (non-Hispanic white vs other), ln(apnea-hypopnea index + 1), and the study site from which the subjects were recruited; and 3, adjusted for all variables in model 2 plus waist girth.

likely to be women (55.9%); their mean (SD) BMI (28.3 [5.1]) and the distribution of usual sleep time were similar. Diabetes mellitus was present in 20.9% of included subjects, 41.6% of whom were taking antidiabetic medication; IGT was present in an additional 28.2% of subjects. Most subjects reported sleeping 7 to 8 hours per night, although a usual sleep time of 6 hours or less was reported by 27.1%, including 8.4% sleeping 5 hours or less per night. Of the subjects, 8.6% reported sleeping 9 hours or more per night. The mean interval between assessment of sleep time and determination of glycemic status was 5.5 months (range, 0-12 months). Subjects at the extremes of sleep time were somewhat older and more likely to be women or minorities, although this difference was significant only for age (**Table 1**). Obesity measures and AHI were similar across sleep time categories. Overall, 32.3% of subjects reported frequently experiencing at least 1 symptom of insomnia. Subjects reporting shorter sleep times had a significantly higher prevalence of insomnia symptoms (Table 1).

Compared with sleep times of 7 to 8 hours per night, self-reported usual sleep times of 6 or less or 9 or more hours per night were associated with a higher adjusted odds ratio (OR) for IGT and DM (**Table 2**). As expected, older age and greater waist girth were associated with a higher adjusted OR for DM and IGT, while there was a trend toward a higher adjusted OR in women, minorities, and persons with a higher AHI. Adjustment for these variables did not weaken the observed associations of sleep time to DM and IGT (Table 2). There was also little influence on the association of sleep time with DM and IGT when the models were additionally ad-

Table 3. Data for Diabetes Mellitus or Impaired Glucose Tolerance by Reported Usual Sleep Time in Stratified Analyses

Variable	No. of Subjects	Reported Usual Sleep Time per Night, h*			*P* Value†
		≤5	6	≥9	
Sex					
Male	722	1.93 (1.04-3.56)	1.19 (0.79-1.80)	1.91 (1.07-3.42)	.04
Female	764	1.83 (1.07-3.11)	2.18 (1.46-3.24)	1.84 (1.06-3.19)	<.01
Age, y					
<70	690	1.62 (0.87-3.03)	1.70 (1.12-2.58)	1.91 (0.97-3.75)	.02
≥70	796	2.00 (1.20-3.34)	1.58 (1.08-2.33)	1.87 (1.14-3.05)	<.01
AHI					
<5	715	1.95 (1.08-3.50)	1.81 (1.22-2.70)	1.79 (1.02-3.16)	<.01
≥5	771	1.77 (1.03-3.02)	1.45 (0.97-2.17)	1.95 (1.12-3.40)	.02
Insomnia					
Absent	997	1.76 (0.81-3.83)	1.78 (1.22-2.59)	1.89 (1.21-2.96)	<.01
Present	480	1.66 (0.98-2.82)	1.40 (0.88-2.22)	1.56 (0.66-3.70)	.21

Abbreviation: See Table 1.

*Data are given as odds ratios (95% confidence intervals) for the presence of either diabetes mellitus or impaired glucose tolerance relative to normal glucose tolerance, from logistic regression models using 7 to 8 hours of sleep per night as the referent category. Analyses are adjusted for age, age^2, sex, waist girth, race/ethnicity (non-Hispanic white vs other), ln(AHI + 1), and the study site from which the subjects were recruited.

†*P* values reflect the overall significance level of the effect of sleep time on diabetes mellitus and impaired glucose tolerance within strata, based on the likelihood ratio χ^2_8; there was no significant interaction of sleep time with sex, age, AHI, or presence of insomnia.

justed for systolic blood pressure, use of antihypertensive medications, prevalent coronary artery disease, heart failure or stroke, current cigarette smoking, usual daily consumption of caffeine or alcohol, and frequency of depressive symptoms, nocturia, nocturnal leg jerks or cramps, and nocturnal joint pain. There was little difference between sleep times of 7 and 8 hours in the adjusted ORs for DM and IGT; comparing 7 with 8 hours of sleep, the adjusted ORs were 0.92 (95% confidence interval, 0.64-1.32) and 1.03 (95% confidence interval, 0.75-1.40) for DM and IGT, respectively. When subjects with DM were excluded from the analysis, the association of sleep time with IGT was essentially unchanged from the model presented in Table 2, and remained statistically significant ($P<.005$).

Secondary analyses demonstrated that the association of sleep time with impaired glucose regulation was significant in men and women, in subjects 70 years and older and younger than 70 years, and in those with and without obstructive sleep apnea/hypopnea, defined as an AHI of 5 or more (**Table 3**), with no significant effect modification by these factors. None of the insomnia measures examined was significantly associated with glycemic status; therefore, despite their strong association with short sleep time, their inclusion in the regression model had no meaningful effect on the association of sleep time to IGT and DM. When stratified on the presence or absence of insomnia, the adjusted OR for DM or IGT among short and long sleepers was similar in those with and without insomnia (Table 3). Moreover, the association of sleep time with glycemic status remained significant when the analysis was restricted to the 997 subjects without insomnia symptoms.

COMMENT

By using uniformly applied objective measures to assess glycemic status, the present study provides epidemiologic evidence that short sleep time is associated with DM and IGT in community-dwelling middle-aged and older adults under conditions of sleep deprivation that are highly prevalent in the United States and other industrialized societies. This finding persisted after adjustment for known DM risk factors, and was independent of the presence of insomnia symptoms, suggesting that voluntary sleep restriction may be a cause of impaired glucose regulation. The association of short sleep times with DM and IGT may explain in part the association between short sleep time and myocardial infarction[16] and mortality,[4-7] and lends empirical support to the common recommendation to obtain 7 to 8 hours of sleep per night. Moreover, it suggests that obtaining an adequate total sleep time should be tested as a nonpharmacologic treatment modality in the management of patients with DM and IGT.

These results are consistent with a prior report from the Nurses' Health Study in which the adjusted ORs for incident DM over a mean follow-up of 10 years were 1.18, 1.10, and 1.29 in subjects sleeping 5 or less, 6, or 9 or more hours per night, respectively, compared with those sleeping 8 hours per night.[17] Although the association of sleep time with incident DM in the Nurses' Health Study was significant only in the subset of diabetic patients with severe symptoms, and not in the entire cohort, the power of that study may have been limited by use of self-report to identify incident cases of DM or by changes in sleep habits during the long follow-up period. A study using an intravenous glucose tolerance test in a small sample of healthy, nonobese, young adults with habitual short sleep times (mean, 5.3 hours per night) found that, compared with subjects sleeping 7.5 to 8.5 hours per night, short sleepers were not glucose intolerant but did have reduced insulin sensitivity.[18] Perhaps with additional risk factors, such as advancing age or greater adiposity, this reduced insulin sensitivity would result in glucose intolerance.

In this study, we have adjusted for factors believed a priori to be potential confounders of the association between sleep time and glycemic status, including age, sex, race, and obesity, and for AHI, given the growing evi-

dence that obstructive sleep apnea/hypopnea may cause impaired glucose regulation.[19,20] Waist girth was used to measure obesity, because it correlates better than either BMI or waist-hip ratio with visceral adiposity,[21] which is more relevant than total body fat to the pathophysiological features of DM.[22,23] In contrast to the Nurses' Health Study report,[17] adjustment for obesity did not attenuate the association of sleep time with DM, implying that confounding by adiposity is unlikely. While it has been suggested that sleep restriction may lead to obesity by suppressing leptin secretion,[24] we did not observe greater obesity in short sleepers, in contrast to the modest association of short sleep with obesity in prior reports.[7,17] This may reflect the younger age of subjects in those studies, because the association of sleep time with obesity is reported to diminish with age.[25] Secondary analyses also indicate that the association of sleep time with DM and IGT was not confounded by caffeine or alcohol consumption, cigarette smoking, or antihypertensive medication use, which might influence sleep habits, or nocturia or prevalent medical illnesses often associated with DM, including hypertension and cardiovascular disease.

Studies of experimental sleep restriction suggest a likely causal association between short sleep and impaired glucose regulation. Sleep restriction to 4 hours per night for 6 nights caused IGT in healthy young adults, which resolved after 1 week of increased sleep duration.[8] The biological mechanisms underlying this effect are uncertain. Sleep deprivation may lead to increased sympathetic nervous system activity,[8,26] which may impair glucose regulation via the lipolytic effects of β-adrenergic stimulation of visceral adipose tissue.[27] Sleep deprivation also alters activity of the hypothalamic-pituitary-adrenal axis, with short-term partial sleep deprivation causing a shorter quiescent period of cortisol secretion and slower clearance of free cortisol.[8,28] Experimentally delaying sleep onset is associated with a presleep burst of growth hormone secretion followed by the usual sleep-onset growth hormone secretion,[29] possibly causing morning glucose intolerance, although persistence of this pattern of growth hormone secretion with long-term delayed sleep onset is uncertain. Primary insomnia is associated with increased activity of the hypothalamic-pituitary-adrenal axis,[30,31] and patients with insomnia often underestimate their actual sleep time.[32] However, the observed association of short sleep time with DM and IGT in the present study remained significant after adjustment for insomnia or excluding subjects with insomnia, implying that voluntary sleep restriction at levels common in the population may lead to impaired glucose regulation.

The mechanisms mediating the association of long sleep time with impaired glucose regulation are more speculative. Seven days of extending time in bed to 12 hours per night was not associated with evidence of glucose intolerance.[8] Nurses' Health Study subjects who reported sleeping 9 hours or more per night reported 15% less physical activity per week than those sleeping 7 to 8 hours per night.[17] This might lead to impaired glucose regulation through direct effects of inactivity[33,34] or through an association of inactivity with a greater degree of visceral adiposity for a given level of total body adiposity, as suggested by the greater reduction in visceral compared with total body fat with daily walking.[35] Depression is associated with increased cortisol level and may cause increased sleep time. Although depression was not formally assessed in our subjects, sleep time was not significantly associated with depressive symptoms obtained from the Medical Outcomes Study 36-Item Short-Form Health Survey; confounding by depression is, therefore, unlikely. While adjustment for usual alcohol consumption did not meaningfully alter the association of sleep time with DM or IGT, sleep time was significantly associated with alcohol consumption. Because heavy alcohol users may underreport their actual consumption, it is possible that alcohol use contributes to the higher prevalence of impaired glucose regulation in those sleeping 9 hours or more per night. An alternative hypothesis is that conditions associated with mild chronic inflammation, such as subclinical cardiovascular disease or visceral obesity per se, cause long sleep time and alteration in glycemic control via the sleep-inducing[36,37] and metabolic[38,39] effects of inflammatory cytokines, including interleukin 1 and tumor necrosis factor α.

Several limitations of this study merit discussion. Usual sleep time was obtained by self-report. Although actigraphic studies have demonstrated the validity of self-reported sleep time,[40] it does have a moderate degree of variation over time. While some misclassification on sleep time is likely, this should bias the study toward a null result. Because this was an observational study, the possibility of confounding by unmeasured variables, such as sedentary lifestyle and diet, cannot be excluded. In the Nurses' Health Study, however, the mean level of physical activity was nearly identical in those sleeping 6 hours or less and those sleeping 7 to 8 hours per night.[17] Because this was a cross-sectional study, the temporal relation between sleep time and impaired glucose regulation is unknown. Diabetes mellitus and IGT might affect sleep time, because diabetic patients may experience nocturia, neuropathic pain, or restless legs syndrome, which impairs sleep initiation or maintenance. We found no significant association, however, of DM or IGT with insomnia symptoms or frequency of nocturia, nocturnal leg jerks or cramps, or nocturnal joint pain. Finally, this was a study of adults aged 53 to 93 years. Although the relation of sleep time to DM and IGT was similar in those 70 years and older and in those younger than 70 years, caution must be exercised in extrapolating these findings to younger age groups.

Accepted for Publication: October 13, 2004.
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Funding/Support: This study was supported by National Heart, Lung and Blood Institute cooperative agreements U01 HL53940 (University of Washington, Seattle), U01 HL53941 (Boston University), U01 HL53938 (University of Arizona, Tucson), U01 HL53916 (University of California, Davis), U01 HL53934 (University of Minnesota, Minneapolis), U01 HL53931 (New York University, New York), U01 HL53937 and U01 HL64360 (The Johns Hopkins University, Baltimore, Md), U01 HL63463 (Case Western Reserve University, Cleveland, Ohio), and U01 HL63429 (Missouri Breaks Industries Research, Inc, Timber Lake, SD), and cooperative agreement supplement HL53938-07S1 (University of Arizona).
Acknowledgment: The SHHS acknowledges the Atherosclerosis Risk in Communities Study, the Cardiovascular Health Study, the Framingham Heart Study, the Cornell/Mt Sinai Worksite and Hypertension Studies, the Strong Heart Study, the Tucson Epidemiologic Study of Airways Obstructive Diseases, and the Tucson Health and Environment Study for allowing their cohort members to be part of the SHHS and for permitting data acquired by them to be used in the study. The SHHS is particularly grateful to the members of these cohorts who agreed to participate in the SHHS as well. The SHHS further recognizes all of the investigators and staff who have contributed to its success. A list of SHHS investigators, staff, and their participating institutions is available on the SHHS Web site (http://www.jhsph.edu/shhs).

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