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Short Sleep Duration as a Risk Factor for Hypertension Analyses of the First National Health and Nutrition Examination Survey

James E. Gangwisch, Steven B. Heymsfield, Bernadette Boden-Albala, Ruud M. Buijs, Felix Kreier, Thomas G. Pickering, Andrew G. Rundle, Gary K. Zammit, Dolores Malaspina

Abstract—Depriving healthy subjects of sleep has been shown to acutely increase blood pressure and sympathetic nervous system activity. Prolonged short sleep durations could lead to hypertension through extended exposure to raised 24-hour blood pressure and heart rate, elevated sympathetic nervous system activity, and increased salt retention. Such forces could lead to structural adaptations and the entrainment of the cardiovascular system to operate at an elevated pressure equilibrium. Sleep disorders are associated with cardiovascular disease, but we are not aware of any published prospective population studies that have shown a link between short sleep duration and the incidence of hypertension in subjects without apparent sleep disorders. We assessed whether short sleep duration would increase the risk for hypertension incidence by conducting longitudinal analyses of the first National Health and Nutrition Examination Survey ($n=4810$) using Cox proportional hazards models and controlling for covariates. Hypertension incidence ($n=647$) was determined by physician diagnosis, hospital record, or cause of death over the 8- to 10-year follow-up period between 1982 and 1992. Sleep durations of ≤ 5 hours per night were associated with a significantly increased risk of hypertension (hazard ratio, 2.10; 95% CI, 1.58 to 2.79) in subjects between the ages of 32 and 59 years, and controlling for the potential confounding variables only partially attenuated this relationship. The increased risk continued to be significant after controlling for obesity and diabetes, which was consistent with the hypothesis that these variables would act as partial mediators. Short sleep duration could, therefore, be a significant risk factor for hypertension. (*Hypertension*. 2006;47:833-839.)

Key Words: circadian rhythm ■ obesity ■ diabetes mellitus ■ hypertension, essential ■ sleep

The prevalence of hypertension has increased over the past decade despite improvements in awareness, treatment, and control of the disease.¹ During this same time period, the average sleep duration in the United States has steadily declined and has been fueled by increased opportunities to engage in activities that compete for the time allotted to sleep.² There is evidence to suggest that the increasing prevalence of hypertension and the decline in average sleep time are related. Sleep deprivation studies of both normotensive^{3,4} and hypertensive⁵ subjects have shown significant increases in blood pressure and sympathetic nervous system activity after nights where sleep was restricted to 3.6 to 4.5 hours. Long-term treatment with melatonin, the night hormone that promotes sleep, has been shown to reduce blood pressure in hypertensive subjects.⁶ Short sleep duration also has been shown to increase the risk for the incidence of myocardial infarction in women, possibly as a consequence of increased blood pressure.⁷

Prolonged exposure to short sleep durations could function to develop and maintain hypertension and its vascular and cardiac complications. Blood pressure and heart rate follow a diurnal pattern with the lowest values occurring during sleep. Blood pressure gradually falls with the onset of sleep and then remains low until the moment of awakening, when it promptly rises.⁸ While normotensive subjects sleep, their blood pressure dips by an average 10% to 20%,⁹ whereas in hypertensive subjects, this diurnal profile is generally preserved but at a higher level of blood pressure.¹⁰ Sleeping fewer hours per night would, therefore, function to raise average 24-hour blood pressure and heart rate. Shorter sleep durations would also result in longer exposures to elevated sympathetic nervous system activity and to waking physical and psychosocial stressors. Increased exposure to stress has been shown to promote salt appetite and suppress renal salt-fluid excretion.¹¹ Long-term exposure to increased total 24-hour hemodynamic load associated with short sleep dura-

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tions could lead to structural adaptations, such as arterial and left ventricular hypertrophic remodeling, that gradually reset the entire cardiovascular system to operate at an elevated pressure equilibrium.¹²

Sleep apnea has long been known to be associated with hypertension and cardiovascular disease. Obesity accounts for a significant proportion of this association, because it is highly comorbid with sleep apnea and is a significant risk factor for hypertension.¹³ However, we are not aware of any published prospective population studies that have shown a link between short sleep duration and the incidence of hypertension in subjects without apparent sleep disorders.

In this study, we examined the relationship between self-reported sleep duration and the diagnosis of hypertension during 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).^{14–16} We hypothesized that prolonged short sleep duration is associated with hypertension. We theorized that obesity and diabetes act as partial mediators of this relationship, because short sleep duration is associated with obesity^{17,18} and the incidence of type 2 diabetes¹⁹ and because depriving healthy subjects of sleep has been shown to decrease leptin, increase ghrelin, increase appetite,²⁰ and compromise insulin sensitivity.²¹ We also hypothesized that the relationship between short sleep duration and the incidence of hypertension is stronger in younger subjects than in older subjects, because stronger associations have been found in younger populations than in older populations between sleep-disordered breathing and hypertension²² and between short sleep duration and obesity.^{17,18}

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 baseline measures of self-reported sleep duration were taken from the 1982–1984 survey, and then hypertension incidence was determined over an 8- to 10-year period until 1992. The NHANES I survey was a probability sample of the civilian noninstitutionalized population of the United States between 1971 and 1975. The survey included a standardized medical examination and questionnaires to obtain information on the effects of clinical, environmental, and behavioral factors on health conditions. The NHANES I epidemiologic follow-up study conducted between 1982 and 1984 attempted to trace and interview NHANES I subjects, or their proxies, who were aged 25 to 74 years at baseline ($n=14\,407$). Of all eligible subjects, 85% were successfully recontacted ($n=12\,220$). In the 1982–1984 follow-up survey, subjects were 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 ($n=734$), who had missing data on any of the covariates ($n=149$), and who had hypertension at or before the 1982–1984 survey ($n=4830$) were excluded from the analyses, yielding a final sample size of 4810 subjects. This study involved analyses of a publicly available data set that did not include identifying information and, therefore, met federal guidelines for exemption from institutional review board review.

Assessment of Hypertension

Individuals who had systolic blood pressure readings >140 mm Hg or diastolic readings >90 mm Hg at the time of the 1982–1984 survey or who had been diagnosed with hypertension at or before the

1982–1984 survey were excluded from the analyses. The blood pressure measurement procedures used in the NHANES I were adapted from those of the American Heart Association and Hypertension Detection and Follow-Up Program.²³ Three blood pressure readings were taken at the same sitting, and readings were not taken if maximum inflation level was >160 mm Hg. Incident cases of hypertension over the 8- to 10-year follow-up period between 1982 and 1992 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.

Assessment of Covariates

The 1982–1984 questionnaire included questions on history of diabetes, age, physical activity, alcohol consumption, salt consumption, pulse rate, daytime sleepiness, depression, smoking, education, ethnicity, and gender. We measured salt consumption by adding together the subject's responses to 2 questions asking them to estimate how frequently they use salt at the table and how often they eat salty snacks. To measure the presence of depressive symptoms, we used the standard cutoff score of 16 from a total possible score of 60 on the 20-question Center for Epidemiologic Studies Depression Scale (CES-D). Subjects who did not answer any of the CES-D questions were excluded from the analyses. A total of 183 subjects (3.8% of the sample) had missing data on ≥ 1 of the 20 CES-D questions. Missing values for specific CES-D questions were imputed with the mean of the questions that the subject did answer. To compute body mass index (BMI; kg/m^2), we used heights from medical examinations conducted between 1971 and 1975 and actual body weights measured with scales at the 1982–1984 interviews. A total of 75 subjects (1.6% of subjects) had missing values for measured body weight, but for 70 of these subjects, we were able to substitute their measured body weight with their self-reported body weight. The measured and self-reported body weights obtained in 1982–1984 for the entire sample had a Pearson correlation coefficient of 0.975, indicating a reasonable level of accuracy for the self-reported weights.

Statistical Analyses

After preliminary univariate and bivariate analyses, we used Cox proportional hazards models to examine the effect of sleep duration on the risk of being diagnosed with hypertension over the 8- to 10-year follow-up period. The time duration to diagnosis was determined from the baseline date to the first report of hypertension. Covariates in the first adjusted multivariate model (model 2) included daytime sleepiness (never/rarely/sometimes or often/almost always), depression (yes or no), physical activity (6=high, 5, 4, 3, 2=low), alcohol consumption (0, >0 and <28 , or ≥ 28 g/day), salt per day (continuous), current smoking status (0, 1 to 5, 6 to 10, 11 to 20, or >20 cigarettes per day), pulse rate, and gender. We chose to include the demographic variables of education (high school graduate or $>$ high school graduate), age (5-year interval), and ethnicity (white or nonwhite, including black, Hispanic, Asian, and other) in a separate model (model 3), because we hypothesized that a significant proportion of their ability to influence the results would be based on the fact that they are associated with being overweight and obese. We included BMI (lean= <25 , overweight= >25 and <30 , or obese= ≥ 30) and history of diabetes (yes or no) in the final model (model 4) to test whether these variables acted as partial mediators of the relationship between sleep duration and the incidence of hypertension. The significance of individual coefficients in the Cox proportional hazards models were determined by the 95% confidence limits for hazards ratios (HRs).

We tested for multiplicative interaction between age and duration of sleep with the log likelihood ratio test and found that age acted as an effect modifier between the number of hours of sleep per night and the incidence of hypertension ($P<0.05$). We then stratified the sample by 10-year age increments and performed Cox proportional hazards analyses with each. The HRs for the incidence of hypertension for sleeping ≥ 7 hours per night compared with the other sleep categories were similar for subjects who were in their 30s, 40s, and 50s at the time of the 1982–1984 follow-up. We chose to divide the

TABLE 1. Relationship Between Sleep Duration at Baseline and Diagnosis of Hypertension Over Follow-Up

Hours of Sleep	Diagnosed With Hypertension Over Follow-Up, n (%)	Not Diagnosed With Hypertension Over Follow-Up, n (%)	χ^2 (P)
Ages 32 to 86 y (n=4810)			28.84 (P=0.0001)
≤5 h (n=358)	74 (12)	284 (7)	
6 h (n=931)	128 (20)	803 (19)	
7 to 8 h (n=3173)	391 (60)	2,782 (67)	
≥9 h (n=348)	54 (8)	294 (7)	
Ages 32 to 59 y (n=3620)			28.00 (P<0.0001)
≤5 h (n=241)	57 (12)	184 (6)	
6 h (n=730)	102 (22)	628 (20)	
7 to 8 h (n=2451)	290 (61)	2,161 (69)	
≥9 h (n=198)	23 (5)	175 (5)	
Ages 60 to 86 y (n=1190)			5.05 (P=0.1682)
≤5 h (n=117)	17 (10)	100 (10)	
6 h (n=201)	26 (15)	175 (17)	
7 to 8 h (n=722)	101 (57)	621 (61)	
≥9 h (n=150)	31 (18)	119 (12)	

sample into 2 age groups with subjects who, at the time of the 1982–1984 study, were between the ages of 32 and 59 years in one group and subjects who were between the ages of 60 and 86 years in another group. After excluding subjects who were deceased, who did not answer the sleep duration question, who had missing data on any of the covariates, and who had hypertension at or before the 1982–1984 survey, there were 3620 subjects between the ages of 32 and 59 years and 1190 subjects between the ages of 60 and 86 years for the analyses.

The NHANES I included weights to account for the complex sampling design and to allow approximations of the US population. We conducted nonweighted analyses using SAS software²⁴ for 3 reasons. First, our objective was not to provide national estimates but to look at the relationship between sleep duration and the incidence of hypertension. Second, the baseline measures of our study were taken from the 1982–1984 follow-up to the NHANES I, so the

weights created for baseline measures taken from the 1971–1974 NHANES I did not account for subjects who were lost to follow-up between the 2 waves. Third, there have been differences of opinion regarding the appropriateness of using the sample weights with the NHANES.²⁵

Results

Results for the bivariate analyses at baseline are shown in Tables 1 and 2. There were significant differences between sleep duration categories and the incidence of hypertension for the total sample of subjects between the ages of 32 and 86 years. The relationships between sleep duration categories and the incidence of hypertension were quite different, however, between the younger subjects between the ages of

TABLE 2. Baseline Characteristics and Risk Factors for Hypertension by Self-Reported Sleep Duration

Baseline Characteristics and Risk Factors for Hypertension	Hours of Sleep			
	≤5	6	7 to 8	≥9
n (%)	358 (7.4)	931 (19.4)	3,173 (66.0)	348 (7.2)
Mean (SD)				
Age, y	54.1 (13.7)	50.3 (12.2)	50.5 (12.7)	57.0 (16.3)
BMI, kg/m ²	26.1 (4.4)	25.7 (4.6)	25.2 (4.4)	25.3 (4.8)
Physical activity score	3.9 (1.2)	4.1 (1.1)	4.1 (1.1)	3.8 (1.1)
Alcohol consumption, g/day	6.3 (13.6)	7.8 (16.8)	7.6 (16.0)	8.2 (21.2)
Salt consumption per day	0.91 (1.0)	0.98 (1.0)	0.94 (1.0)	0.88 (1.1)
Pulse rate	71.7 (9.7)	70.3 (9.8)	70.1 (9.8)	71.1 (10.8)
% of subjects				
Diabetes	6	4	3	4
Depression	30	15	12	16
Daytime sleepiness	20	14	11	20
Women	65	62	64	64
Non-white	15	12	8	12
High school graduate	58	71	74	58

32 and 59 years and the older subjects between the ages of 60 and 86 years. For the younger subjects, a higher percentage of those who reported at baseline getting <7 hours of sleep per night were diagnosed with hypertension over the follow-up period than those who reported sleeping 7 to 8 hours per night. For the older subjects, a higher percentage of those who reported getting ≥ 9 hours of sleep per night were diagnosed with hypertension over the follow-up period than those who reported sleeping 7 to 8 hours per night. Table 2 shows the baseline characteristics of the entire sample according to their sleep duration categories. In comparison to self-reported sleep durations of 7 or 8 hours, sleep durations of ≤ 5 hours were associated with older age, higher BMI, lower physical activity, lower alcohol consumption, higher pulse rate, diagnosis of diabetes, depression, daytime sleepiness, nonwhite ethnicity, and less than a high school graduate education. Sleep durations of ≥ 9 hours were associated with older age, lower physical activity, higher alcohol consumption, lower salt intake, higher pulse rate, depression, daytime sleepiness, nonwhite ethnicity, and less than a high school graduate education.

Table 3 shows the HRs of being diagnosed with hypertension over the 8- to 10-year follow-up period as computed with Cox proportional hazards models. There were 647 total incident cases of hypertension over this period, 472 of which occurred in subjects between the ages of 32 and 59 years and 175 of which occurred in subjects between the ages of 60 and 86 years. The test of significance for the interaction between age category and sleep duration was significant ($P=0.0065$). In the unadjusted results (model 1) for the total sample between the ages of 32 and 86, subjects who reported sleeping ≤ 5 hours per night were significantly more likely (HR, 1.76; 95% CI, 1.37 to 2.56) to have been diagnosed with

hypertension over the follow-up period than subjects who reported getting 7 or 8 hours of sleep per night. However, these results varied by age, with younger subjects who slept ≤ 5 hours per night being over twice as likely (HR, 2.10; 95% CI, 1.58 to 2.79) to be diagnosed with hypertension and older subjects who slept ≥ 9 hours per night being significantly more likely (HR, 1.54; 95% CI, 1.03 to 2.30) to be diagnosed with hypertension. After adjusting for daytime sleepiness, depression, physical activity, alcohol consumption, salt consumption, smoking, pulse rate, and gender (model 2), those who slept ≤ 5 hours per night continued to be significantly more likely (HR, 1.51; 95% CI, 1.17 to 1.95) to have been diagnosed with hypertension. The addition of demographic variables that are associated with obesity (education, age, and ethnicity) in model 3 attenuated the relationship. Consistent with our hypothesis that BMI and history of diabetes would act as partial mediators of the relationship between sleep duration and the incidence of hypertension, the addition of these variables in model 4 further attenuated the results. The total sample of subjects who reported getting ≤ 5 hours of sleep per night continued to be significantly more likely (HR, 1.32; 95% CI, 1.02 to 1.71) to be diagnosed with hypertension after controlling for obesity, history of diabetes, and the other covariates. However, the results differ between the younger and older subjects. Subjects between the ages of 32 and 59 years who reported sleeping ≤ 5 hours per night were significantly more likely (HR, 1.60; 95% CI, 1.19 to 2.14) to have been diagnosed with hypertension than subjects who reported getting 7 or 8 hours of sleep per night, whereas subjects between the ages of 60 and 86 years who slept ≤ 5 hours were actually slightly less likely (HR, 0.85; 95% CI, 0.50 to 1.45), although not significantly so, to have been diagnosed with hypertension.

TABLE 3. HRs (95% CI) of Hypertension Incidence Over the Follow-Up Period by Sleep Duration at Baseline

Hours of Sleep	Model 1*	Model 2†	Model 3‡	Model 4§
Ages 32 to 86 y				
≤ 5 h	1.76 (1.37 to 2.56)	1.51 (1.17 to 1.95)	1.44 (1.11 to 1.85)	1.32 (1.02 to 1.71)
6 h	1.11 (0.91 to 1.35)	1.07 (0.88 to 1.31)	1.06 (0.87 to 1.29)	1.01 (0.82 to 1.23)
7 to 8 h	1.00	1.00	1.00	1.00
≥ 9 h	1.32 (0.99 to 1.75)	1.18 (0.88 to 1.57)	1.13 (0.85 to 1.51)	1.12 (0.84 to 1.50)
Ages 32 to 59 y				
≤ 5 h	2.10 (1.58 to 2.79)	1.84 (1.38 to 2.46)	1.74 (1.30 to 2.32)	1.60 (1.19 to 2.14)
6 h	1.18 (0.94 to 1.48)	1.14 (0.91 to 1.43)	1.13 (0.90 to 1.41)	1.05 (0.83 to 1.31)
7 to 8 h	1.00	1.00	1.00	1.00
≥ 9 h	0.98 (0.64 to 1.50)	0.91 (0.59 to 1.39)	0.91 (0.59 to 1.40)	0.92 (0.60 to 1.41)
Ages 60 to 86 y				
≤ 5 h	1.05 (0.63 to 1.75)	0.86 (0.51 to 1.46)	0.86 (0.51 to 1.47)	0.85 (0.50 to 1.45)
6 h	0.90 (0.58 to 1.38)	0.88 (0.57 to 1.36)	0.85 (0.55 to 1.32)	0.86 (0.56 to 1.33)
7 to 8 h	1.00	1.00	1.00	1.00
≥ 9 h	1.54 (1.03 to 2.30)	1.36 (0.90 to 2.06)	1.32 (0.87 to 2.01)	1.31 (0.86 to 1.99)

*Model 1, unadjusted.

†Model 2, adjusted for daytime sleepiness, depression, physical activity, alcohol consumption, salt consumption, smoking, pulse rate, and gender.

‡Model 3, adjusted for the variables in model 2 plus education, age, and ethnicity.

§Model 4, adjusted for the variables in model 3 plus overweight/obesity and diabetes.

Discussion

We observed an association between short self-reported sleep duration and the incident diagnosis of hypertension in a large US sample. Subjects between the ages of 32 and 59 years at baseline who reported averaging ≤ 5 hours of sleep per night were at an increased risk for developing hypertension over the follow-up period. An association between short sleep duration and hypertension incidence was not found for subjects who were between the ages of 60 and 86 years at baseline. There are a number of factors that may explain the different relationships found between short sleep duration and the incidence of hypertension in the younger and older age groups. First, subjects experiencing hypertension, obesity, and diabetes would be less likely to survive into their later years. Second, advanced age is associated with changes in sleep architecture with increased difficulties in sleep initiation and maintenance.²⁶ Elderly subjects, who are often retired, also have more opportunities to take naps during the day. Third, sleep disordered breathing is associated with hypertension in subjects below the age of 60 years but not in subjects 60 years of age or older.²² Activation of the sympathetic nervous system is an important mechanism that links sleep-disordered breathing to combined systolic and diastolic hypertension, which is common in middle aged hypertensive patients. However, no mechanistic link has been identified between sleep-disordered breathing and isolated systolic hypertension that results from age-dependent loss of arterial compliance and accounts for nearly 60% of hypertension in elderly populations.²² Fourth, short sleep duration has been shown to be more strongly associated with obesity in younger subjects than in older subjects.^{17,18} Fifth, because the sample size of older subjects was low in comparison to the sample size for younger subjects, the lack of an association between short sleep duration and hypertension incidence in the older subjects could be because of a lack of statistical power and, therefore, could represent a type II error.

The association between short sleep duration and the incidence of hypertension was attenuated by the inclusion in the multivariate models of diabetes, body weight, and covariates highly correlated with body weight, such as education, age, and ethnicity. The effect of short sleep duration on hypertension incidence is, therefore, likely to be partially related to the influence of short sleep duration on body weight, perhaps by influencing leptin, ghrelin, appetite, and insulin sensitivity.

The association between short sleep duration and hypertension incidence continued to be statistically significant after controlling for body weight. Short sleep duration is, therefore, likely to have direct effects on the risk for the incidence of hypertension independent of its influence on body weight. These results are consistent with physiological data from previous studies on the effects of sleep deprivation on normotensive^{3,4} and hypertensive⁵ subjects. In 2 of those studies, on days after nights of sleep deprivation, significant increases in blood pressure were accompanied by increases in the urinary excretion of norepinephrine indicative of increased sympathetic nervous system activity.^{3,5} These findings have led to the hypothesis that the mechanism by which sleep deprivation, a stressful condition, raises blood pressure

is by increasing the synthesis of catecholamines through the activation of superior centers.⁵ In all 3 of the studies on the effects of sleep deprivation on blood pressure, subjects had a regular sleep-wake schedule (≈ 8 hours per night) before their sleep was restricted to 3.6 to 4.5 hours per night. The sleep durations of the subjects in our study were not necessarily restricted from a previously regular sleep-wake schedule, so their experience of short sleep duration was likely to have been qualitatively and physiologically different from those of the subjects in the sleep deprivation studies. We also do not know whether short self-reported sleep durations in the NHANES I were indicative of sleep deprivation or whether they simply represented the normal variability between individuals in required sleep durations.

Habitually short sleep durations could lead to the development and maintenance of hypertension through prolonged exposure to raised 24-hour blood pressure and heart rate, elevated sympathetic nervous system activity, waking physical and psychosocial stressors, and increased salt retention. According to Folkow's¹² hypothesis, extended exposure to these forces could lead to the entrainment of the cardiovascular system to operate at an elevated pressure equilibrium through structural adaptations, such as arterial and left ventricle hypertrophic remodeling.

Chronic short sleep durations could also contribute to hypertension by disrupting circadian rhythmicity and autonomic balance. The central biological clock or suprachiasmatic nucleus (SCN) evolved to synchronize activity and rest to the rising and setting of the sun using hormones and the autonomic nervous system. To generate and organize autonomic rhythms, the SCN requires repeated metabolic cues from light exposure, sleep, activity, and feeding. Dramatic alterations in these parameters in modern industrialized society are theorized to cause the environment sensed by the brain to become metabolically flattened and arrhythmic, disturbing the circadian rhythmicity of blood pressure in susceptible individuals.²⁷ Hypertension is characterized by a disturbance in the circadian rhythmicity of many physiological variables, such as a shifting of the daily blood pressure profile to higher values, an increased prevalence of the nondipping pattern, increased blood pressure variability,¹¹ and disturbances in the diurnal rhythm of cardiac output.²⁸ Hypertensive subjects have been shown to have reductions of $>50\%$ in the 3 main neuronal populations of the SCN in comparisons between normal subjects and hypertensive subjects who died of myocardial infarction or brain hemorrhage.²⁹

Short sleep duration could also influence hypertension incidence by making it more difficult to maintain a healthy lifestyle. In results from the National Sleep Foundation 2002 Sleep in America Poll, not getting enough sleep was associated with irritability, impatience, pessimism, and feeling tired and stressed.² It would seem that these feelings and emotional states would function to lessen one's resolve and willpower to follow dietary or exercise regimens that would be protective against hypertension.

Whereas the results from this epidemiological study lend support to the hypothesis that short sleep duration could lead to hypertension, other important considerations are whether reverse causation contributed toward this finding or whether

some uncontrolled confounder played a part in the results. We cannot rule out the possibility that short sleep duration may be a prodromal symptom of hypertension that predates diagnosis. Sleep-disordered breathing and the related clinical syndrome of sleep apnea have been shown in a prospective study to increase the risk for hypertension incidence,³⁰ so the presence of these disorders could have played a part in the association between short sleep duration and hypertension incidence. The NHANES I follow-up survey did not include questions on sleep disorders, but we would expect that individuals with sleep apnea would be more likely to self-report higher average sleep times, because they are often unaware of their disrupted sleep patterns and require longer sleep durations to compensate for poor sleep quality.

One limitation of this study was the use of self-reported sleep durations as opposed to measured sleep durations. However, good agreement has been found in previous studies between self-reported sleep durations and those obtained through actigraphic monitoring.^{31,32} 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 hypertension incidence. However, investigators from the Nurses' Health Study used a sleep duration question similar to the one used in the NHANES I, and they found good reproducibility of their sleep duration question over 2 years.³³ The question used in the Nurses Health Study asked, "How many hours of actual sleep do you get in a 24-hour period?" We also want to keep in mind that short sleep duration does not equal insomnia and that we do not know whether short sleepers chose to sleep these hours.

Another limitation of this study relates to the fact that hypertension frequently goes undiagnosed. In an analysis of data from the third NHANES, >30% of subjects who were found to experience hypertension were unaware of their condition.¹ We have no way of knowing whether subjects with short sleep durations were more or less likely than subjects with long sleep durations to seek or receive treatment and, therefore, to be diagnosed with hypertension. Other limitations include possible bias arising from loss to follow-up and missing data on baseline risk variables.

Perspectives

The results from this study suggest that short sleep duration could play a role in the etiology of hypertension in some individuals. Further research is needed to investigate the biological mechanisms that link short sleep duration and high blood pressure. If short sleep duration functions to increase blood pressure, then interventions that increase the amount and improve the quality of sleep could potentially serve as treatments and as primary preventative measures for hypertension. Examples of behavioral interventions include helping patients to modify maladaptive sleep habits and educating them about healthier sleep hygiene practices. Studies exploring the efficacy of sleep interventions for the treatment and prevention of high blood pressure could help clarify the

proportion of the hypertension risk attributable to short sleep duration.

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