Insomnia with Objective Short Sleep Duration is Associated with a High Risk for Hypertension

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Study Objectives: To examine the joint effect of insomnia and objective short sleep duration on hypertension risk.

Design: Representative cross-sectional study.

Setting: Sleep laboratory.

Participants: 1,741 men and women randomly selected from central Pennsylvania.

Interventions: None.

Measurements: *Insomnia* was defined by a complaint of insomnia with a duration ≥ 1 year, while *poor sleep* was defined as a complaint of difficulty falling asleep, staying asleep, or early final awakening. Polysomnographic sleep duration was classified into 3 categories: ≥ 6 h sleep (top 50% of the sample); 5-6 h (approximately the third quartile of the sample); and ≤ 5 h (approximately the bottom quartile of the sample). Hypertension was defined based either on blood pressure measures or treatment. We controlled for age, race, sex, body mass index, diabetes, smoking, alcohol use, depression, sleep disordered breathing (SDB), and sampling weight.

Results: Compared to the normal sleeping and > 6 h sleep duration group, the highest risk of hypertension was in insomnia with < 5 h sleep duration group (OR [95% CI] 5.1 [2.2, 11.8]), and the second highest in insomnia who slept 5-6 hours (OR 3.5 [1.6, 7.9] P < 0.01). The risk for hypertension was significantly higher, but of lesser magnitude, in poor sleepers with short sleep duration.

Conclusions: Insomnia with short sleep duration is associated with increased risk of hypertension, to a degree comparable to that of other common sleep disorders, e.g., SDB. Objective sleep duration may predict the severity of chronic insomnia a prevalent condition whose medical impact has been apparently underestimated.

Keywords: Insomnia, objective sleep duration, hypertension

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INSOMNIA IS, BY FAR, THE MOST COMMONLY ENCOUNTERED SLEEP DISORDER IN MEDICAL PRACTICE. HOWEVER, RELATIVELY LITTLE IS KNOWN about the mechanisms, causes, clinical course, and consequences of this highly prevalent chronic condition.[1,2]

Many studies have established that insomnia is highly comorbid with psychiatric disorders and is a risk factor for the development of depression, anxiety, and suicide.[1,2] However, in contrast to the other most common sleep disorder, sleep disordered breathing (SDB), chronic insomnia has not been linked with significant medical morbidity, e.g., cardiovascular disorders. Few studies that have examined the association of chronic insomnia with hypertension have reported modest and inconsistent effects of little or no clinical significance.[3-6] In fact, Kripke et al. found a reduced mortality rate for those individuals complaining of sleep difficulties after 6 years of follow-up.[7]

Most, but not all, studies have reported that insomnia is associated with an overall hypersecretion of ACTH and cortisol, suggesting an activation of the hypothalamic-pituitary-adrenal (HPA) axis in these patients.[8-11] Given the well-established association of hypercortisolemia with significant medical morbidity, e.g., hypertension, metabolic syndrome, osteoporosis,[12] the paucity of data linking insomnia with these medical disorders is a paradox.

In our studies, we observed and reported that the activation of the HPA axis in insomnia was strongly and positively correlated with objective indices of sleep disturbance.[8,9] Specifically, hypercortisolemia was found primarily in insomniacs who demonstrated short sleep duration in the sleep laboratory but not in those whose objective sleep duration was similar to that of normal sleepers. Similarly, earlier studies have shown higher autonomic activation, including heart rate, 24-h metabolic rate, and impaired heart rate variability, in insomniacs selected based on objective polysomnographic criteria.[13-16] Based on these observations, we speculate that objective short sleep duration may be an index of the biological severity of the disorder and that insomniacs with short sleep duration are at high risk for adverse medical outcomes.

To test this hypothesis, we examined the joint effect of the complaints of chronic insomnia and poor sleep (a milder form of insomnia), and objective sleep duration on the prevalent hypertension in a large cross-sectional population-based sample from central Pennsylvania. We hypothesized that chronic insomnia is associated with a significant risk of hypertension, and that the comorbidity of insomnia and hypertension is enhanced by objective short sleep duration.

METHODS

Population

The data presented here were collected as part of a 2-phase protocol whose primary purpose was to establish the age distribution of sleep disordered breathing.[17,18] In the first phase of the study, a sample of adult men and women (age ≥ 20 years) was randomly selected from local telephone households in 2 counties of central Pennsylvania (Dauphin and Lebanon) using the Mitofsky-Waksberg 2-stage random digit dialing procedure.[19] A within-household selection procedure described by Kish was used to select the specific man or woman to be interviewed.[20] Telephone interviews were conducted with 4,364 age-eligible men and 12,219 age-eligible women residing in the sample households for a total sample of 16,583, with a response rate of 73.5% and 74.1%, respectively. The questionnaire employed in this interview included basic demographic and sleep information.

In the second phase of this study, a subsample of 741 men and 1,000 women selected from subjects previously interviewed by telephone were studied in our sleep laboratory. The response rate for this phase was 67.8% and 65.8% for men and women, respectively. We contrasted those subjects who were recorded in the laboratory with those who were selected but not recorded in terms of age, BMI, reported use of antihypertensive medication, and prevalence of sleep disorders. There were no significant differences between these 2 groups on any of these variables. Each subject selected for laboratory evaluation completed a comprehensive sleep history and physical examination. All subjects were evaluated for one night in the sleep laboratory in sound-attenuated, light- and temperature-controlled rooms. During this evaluation, each subject was continuously monitored for 8 h using 16-channel polygraphs including electroencephalogram, electrooculogram, and electromyogram. Bedtimes were adjusted to conform to subjects' usual bedtimes, and subjects were recorded from 2200-2300 to 0600-0700. The sleep records were subsequently scored independently according to standardized criteria.[21] Percent sleep time is total sleep time (duration of sleep) divided by recorded time in bed and multiplied by 100. Respiration was monitored throughout the night by use of thermocouples at the nose and mouth and thoracic strain gauges. All-night recordings of hemoglobin oxygen saturation (SpO_2) were obtained with an oximeter attached to the finger.

Key Measurements

Blood pressure was measured in the evening, about 2 hours before the start of the sleep recording, during the physical examination using a pneumoelectric microprocessor-controlled instrument with the appropriate sized cuffs. The accuracy of this monitor is reported to be ± 3 mm Hg; in addition, internal calibration was performed before each use, and the machine was checked against a mercury sphygmomanometer at least annually. The recorded blood pressure was the average of 3 consecutive readings during a 5-min period following 10 min of rest in the supine position. Hypertension was defined as a diastolic blood pressure > 90 mm Hg or a systolic blood pressure > 140 mm Hg at the time of the sleep laboratory evaluation, or use of antihypertensive medication.

As part of this protocol we assessed the presence of all sleep disorders. The presence of sleep disorders was based on a standardized questionnaire completed by the subjects on the evening of their sleep laboratory visit. This questionnaire consists of 53 questions (7 demographic, 20 sleep-related, and 26 general health questions). In addition, women responded to 8 questions related to menstrual history, menopause, and hormone therapy. Sleep-related questions were qualified in terms of severity on a scale of 0-4 (0 = none, 1 = mild, 2 = moderate, 3 = severe) and duration. Health complaints were also qualified in terms of severity and type of treatment on a scale of 0-7 and duration. The presence of sleep difficulty was established on 3 levels of severity. First, *insomnia* was defined by a complaint of insomnia with a duration of at least 1 year. Second, *poor sleep* was defined as a moderate to severe complaint (based on a mild to severe scale) of difficulty falling asleep, difficulty staying asleep, early final awakening, or unrefreshing sleep. Finally, *normal sleeping* was defined as the absence of either of these 2 categories.

From the objectively recorded sleep time data, we regrouped the entire study sample into 3 ordinal groups: the top 50% of persons (above the median percent sleep time—"normal sleep duration group"); the 25% of persons in the third quartile ("moderately short sleep duration group"); and the bottom 25% of persons ("severely short sleep duration group"). We then rounded the quartile cut-off points to meaningful numbers, and thus created the following 3 sleep duration groups: the "normal sleep duration group," who slept > 6 h; the "moderately short sleep duration group," who slept 5-6 h; and the "severely short sleep duration group," who slept ≤ 5 h.

To control for possible confounding variables influencing the relation between insomnia and hypertension, in the subsample of 1,741 we ascertained whether the respondent was currently treated for depression (including a history of suicidal thoughts or attempts) or diabetes. Diabetes was defined as being treated for diabetes or having a fasting blood sugar > 126 mg/dl from blood drawn the morning after the subject's polysomnogram. Additional information obtained during the polysomnographic evaluation included history of smoking (current use of any type of tobacco product) and alcohol use (> 2 alcohol drinks per day) and objective sleep data including sleep apnea and periodic limb movement assessment. For the purpose of this study, sleep apnea was defined as an obstructive apnea or hypopnea index (OHI) ≥ 5. The condition of periodic limb movement was considered present when there were ≥ 5 movements per hour of sleep. A leg movement was scored when it lasted more than 0.5 seconds, less than 5.0 seconds, and occurred with intervals < 90 seconds between movements (22). Body mass index was based on measured height (cm) and weight (kg) during the subjects' sleep laboratory visit, and data are presented in terms of mean, percentile distribution, and prevalence within each category.

Statistical Analyses

The design of this study included oversampling of those at higher risk for SDB and women with markedly higher BMI to increase the precision of the risk estimates. Because of this

sampling strategy, numeric sampling weights were developed for the analysis so that the estimates could be inferred to the original target population.[17,18,23] Specifically, to improve the efficiency of subject identification and screening, compensatory weights were developed for the analysis to obtain estimates for the original target population of men and women in the 2-county study area. Specifically, 3 weights were created for the men. First, in the telephone sample, 32 of the 963 clusters of phone numbers in the first stage were "exhausted" before the target sample size was obtained. A compensatory weight was computed which corrected for this problem. A second weight was computed because the within-household screening deliberately introduced unequal probabilities of selection across the 3 age groups in order to oversample the middle age group. The final weight for men was computed to account for the oversampling of subjects for the sleep laboratory study (Phase II); those with larger counts of the 4 possible risk factors, i.e., snoring, daytime sleepiness, obesity, and hypertension, had substantially higher probability of being selected. For women, the only weight required was to account for the oversampling of subjects for the sleep laboratory study. To eliminate any suggestion of possible sample bias, we calculated 32 unique weights for the women and 16 unique weights for the men corresponding to all possible combinations of the 5 risk factors for the women (menopause was the fifth risk factor) and 4 for the men. Any individual weight that had a cell size that was too small was combined with adjacent cells, so that < 10% of the cells had a sample < 25, and no cell had a size < 10.[20]

As we began evaluating our sample of women, it appeared that the mean BMI was much higher than that of the national population. Because of the strong association between BMI and sleep apnea, it was felt that a post-stratification population control weight needed to be established to present a more accurate estimate of prevalence. We used the BMI and race distributions by age decade from the NHANES III laboratory data as the standard[23] to adjust both the men and women in terms of BMI and race to be more representative of the national population.

Logistic regression models were used to assess the independent associations of the 3-level sleep difficulty complaints and objective sleep duration with hypertension. In this analysis, hypertension was defined as a diastolic blood pressure > 90 mm Hg or a systolic blood pressure > 140 mm Hg at the time of the sleep laboratory evaluation or the use of antihypertensive medication. The covariables we adjusted for included major confounding factors expected to affect this relation, i.e., age, race, gender, BMI, diabetes, smoking status, alcohol consumption, depression, SDB, and sampling weight. We further tested the interaction between sleep difficulty complaints and objective sleep duration using −2 log likelihood ratio test in logistic regression models. Because of the significant interaction between sleep difficulty complaints and objective sleep duration, we performed final logistic regression models to include 8 dummy variables to represent all 9 possible combinations of sleep difficulty and sleep duration; we used persons with sleep duration > 6 h and without insomnia/poor sleep as a common reference group. We calculated the odds ratios and the 95% confidence intervals (95% CI) from this model to estimate the risk of hypertension associated with different combinations of sleep difficulty complaints and sleep duration, simultaneously adjusting

for age, race, gender, BMI, diabetes, smoking status, alcohol consumption, depression, SDB, and sampling weight.

RESULTS

The demographic, clinical, and sleep characteristics of the entire sample and its subgroups, based on sleep difficulty, the 3 levels of objective sleep duration, and hypertension, are presented in Table 1.

We present the 3 sets of multivariable logistic regression models that examined the association of the 3-level sleep difficulty alone or the 3-level objective sleep duration alone with hypertension after progressively adjusting for potential confounders in Table 2. Insomnia was associated with a significantly higher risk for hypertension that was changed slightly as we increased the number of confounding factors that we adjusted for (OR ranged from 2.76, 95% CI 1.8–4.2, $P < 0.05$, in the first model to OR = 2.41, 95% CI 1.6–3.7, $P < 0.05$, in the last model). Poor sleep was associated with a nonsignificant increase of risk for hypertension (OR, ranged based on number of adjusted variables, from 1.3 to 1.2, and none of the 95% CI excluded 1.00). An objective sleep duration of 5-6 h was associated with a nonsignificant, slight increase of risk for hypertension (OR = 1.1, 95% CI, 0.8–1.5), whereas a sleep duration ≤ 5 h increased the risk by about 50% (OR = 1.56, 95% CI 1.1–2.1, $P < 0.05$) compared with the group that slept > 6 h. In the last model (fully adjusted model), the magnitude of association was much stronger for insomnia than short sleep duration.

The logistic regression results presented in Table 3 examined the joint effect of insomnia and objective sleep duration on hypertension (test of interaction was significant at $P < 0.01$, comparing the model with and without interaction terms). The odds ratios and the 95% confidence intervals presented in Table 3 estimated the risk of hypertension associated with different combinations of sleep difficulty complaints and sleep durations, simultaneously adjusting for age, race, gender, BMI, diabetes, smoking status, alcohol consumption, depression, SDB, and sampling weight. The risk of hypertension was synergistically and significantly increased among persons with both insomnia or poor sleep, and short sleep duration. The presence of both insomnia and an objective sleep duration ≤ 5 h increased the risk for hypertension by about 500% (OR = 5.12., 95% CI 2.2–11.8) compared to the group without insomnia/poor sleep complaint and slept > 6 h. The joint effect of insomnia and a sleep duration of 5-6 h increased the risk for hypertension by about 350% (OR = 3.53, 95% CI 1.6–7.9). For those subjects who complained of poor sleep (milder insomnia) and had sleep duration ≤ 5 h, the joint effect on hypertension was OR = 2.43, 95% CI 1.4–4.3. The association was diminished in subjects with poor sleep and a sleep duration of 5-6 h (OR = 1.48, 95% CI 0.9–2.4). In contrast, in subjects with a complaint of insomnia or poor sleep who slept > 6 h, the risk for hypertension was not significantly increased (OR = 1.31, 95% CI 0.6–2.5 for insomnia, and OR = 0.79, 95% CI 0.5–1.2 for poor sleep, respectively).

Finally, objective short sleep duration in the absence of a sleep complaint was not associated with a significant risk for hypertension. For example, sleep duration of 5-6 h in the absence of a sleep complaint was associated with an OR = 0.86, 95% CI 0.6–1.2. The risk increased slightly in those who slept

Table 1—Demographic, Clinical, and Sleep Characteristics of Study Population

	All	Hypertension		Sleep difficulty			Sleep duration		
		NO	YES	Normal sleeping	Poor sleep	Insomnia	> 6 h	5–6 h	≤ 5 h
	(N = 1741)	(N = 783)	(N = 958)	(N = 1022)	(N = 520)	(N = 199)	(N = 862)	(N = 430)	(N = 449)
Age (Y)	48.7 (13.52)	44.6 (14.01)	56.3 (10.73)	49.3 (14.9)	46.5 (11.56)	49.9 (9.88)	44.01 (12.00)	51.4 (12.11)	58.3 (11.78)
Race (% white)	86	86	86	86	87	76	83	85	93
Sex (% male)	48	45	52	53	37	26	42	50	59
BMI, mean (SD)	27.6 (5.67)	26.5 (6.01)	29.5 (5.01)	27.0 (5.52)	28.8 (5.57)	29.0 (6.11)	27.32 (5.89)	27.8 (5.44)	28.06 (5.40)
BMI (25th, 50th, 75th percentile)	24.0, 26.2, 30.2	23.5, 25.5, 28.6	25.2, 28.3, 32.5	23.8, 26.0, 29.3	24.6, 27.3, 31.9	23.4, 27.7, 32.6	23.8, 26.2, 29.8	24.2, 26.6, 30.3	24.2, 26.5, 30.9
Obesity (%) (BMI ≥ 30)	26	19	39	21	35	40	24	27	29
Diabetes (%)	14	8	25	13	15	18	9	18	22
Hypertension (%)	35	\	\	33	36	52	25	40	56
Hypertension by BP measure (%)	29	\	\	28	31	35	21	32	46
Hypertension by medication (%)	16	\	\	15	17	30	11	18	29
Current smoker (%)	26	28	22	26	27	21	27	21	26
Current alcohol consumption (%)	16	16	17	19	9	6	15	19	16
Depression %	17	16	20	10	30	42	18	17	17
OHI ≥ 5 (%)	11	8	15	11	11	9	7	11	20
Sleep duration (%)									
> 6 hours	56	65	41	72	22	6	N/A	N/A	N/A
5 – 6 hrs	23	21	26	66	26	8	N/A	N/A	N/A
≤ 5 hrs	21	15	34	70	20	11	N/A	N/A	N/A
Sleep difficulty (%)									
Normal sleeping	70	73	65	N/A	N/A	N/A	58	21	21
Poor sleep	22	22	23	N/A	N/A	N/A	55	26	19
Insomnia	8	5	12	N/A	N/A	N/A	44	25	30

All data are adjusted for sampling weight. Where appropriate the standard deviation is presented in parenthesis.

≤ 5 h (OR = 1.13, 95% CI 0.8–1.6). The results remained significant, and the odds ratios very similar to those reported in Table 3 after we adjusted for number of wakes, number of sleep stage changes, percent stage 1 sleep, and periodic limb movements. Further, we reanalyzed the data by defining hypertension based only on recorded blood pressure in the sleep laboratory (diastolic blood pressure > 90 mm Hg or systolic blood pressure > 140 mm Hg). The results remained significant and in the same direction, although, in some instances, the odds ratios reduced toward the null compared to the original analysis. For example, in the group of insomniacs who slept < 5 h, OR = 3.0, 95% CI 1.11–8.09, whereas in the group of poor sleepers who slept < 5 h, OR = 3.3, CI 1.74–6.38.

In summary, insomnia or poor sleep alone, or objective short sleep duration alone may not have a clinically important impact on hypertension risk, but insomnia or poor sleep in combination with objective short sleep duration is associated with a clinically significant risk of hypertension.

DISCUSSION

This is the first study to demonstrate that chronic insomnia associated with objectively measured short sleep duration is a clinically significant risk factor for hypertension. This increased risk is independent of comorbid conditions frequently associated with insomnia or hypertension, such as age, race, obesity, diabetes, alcohol consumption, smoking, sleep disordered breathing, or depression. Furthermore, our findings suggest that objective measures of sleep duration in insomnia may be a useful marker of the biological severity and medical impact of the disorder.

Few studies have assessed the association of insomnia with hypertension, and the results are inconsistent and modest. In a prospective, population-based study that included 8,757 participants, endorsement of either difficulty falling asleep or waking up repeatedly predicted a slightly increased risk of hypertension (OR = 1.2).[3] In that study, the investigators controlled for age, gender, smoking, diabetes, depression, and other comorbid conditions, but did not obtain objective measures of sleep or severity and chronicity of the disorder. In another prospective study from Japan, persistent (> 4 years) complaints of difficulty initiating or maintaining sleep were associated with an increased risk of hypertension (OR = 1.96).[4] The stronger association in this study between insomnia and hypertension may be explained by the addition of severity/chronicity in their definition of insomnia. However, in this study, the potential confounders of sleep disordered breathing and depression were not controlled for.

In our study, the group with the more severe insomnia, i.e., complaint of insomnia ≥ 1 year (with a prevalence of 8% in our general population sample), was associated with a significant risk for hypertension (OR = 2.4). The group with a milder form

Table 2—Multivariable Adjusted Odds Ratio (95% CI) of Hypertension and Insomnia or Objective Sleep Duration

Sleep Difficulty	Model 1			Model 2			Model 3		
	Odds Ratio	95%	CI	Odds Ratio	95%	CI	Odds Ratio	95%	CI
Normal sleeping	1.00			1.00			1.00		
Poor sleep	1.30	0.98	1.72	1.25	0.94	1.70	1.23	0.92	1.65
Insomnia	2.76	1.82	4.20	2.55	1.66	3.90	2.41	1.57	3.70
Sleep duration									
> 6 h	1.00			1.00					
5-6 h	1.19	0.89	1.58	1.18	0.88	1.57	1.13	0.85	1.51
≤ 5 h	1.65	1.22	2.23	1.65	1.22	2.23	1.56	1.14	2.11

Model 1. Adjusted for age, race, sex, BMI, diabetes, and sampling weight.
Model 2. Adjusted for age, race, sex, BMI, diabetes, smoking status, alcohol consumption, depression, SDB, and sampling weight.
Model 3. Adjusted for age, race, sex, BMI, diabetes, smoking status, alcohol consumption, depression, SDB, and sampling weight and objective sleep duration (or insomnia). The interaction between insomnia and objective sleep duration is statistically significant, $P < 0.05$.

Table 3—Multivariable Adjusted Odds Ratio (95% CI) of Hypertension Associated with Insomnia and Objective Sleep Duration

Sleep difficulty	Sleep duration	Sample size	Adjusted OR	95% CI	
Normal sleeping	> 6 h	527	1.00	Low	Upper
Poor sleep	> 6 h	249	0.79	0.52	1.20
Insomnia	> 6 h	86	1.31	0.70	2.46
Normal sleeping	5-6 h	235	0.86	0.60	1.22
Poor sleep	5-6 h	146	1.48	0.90	2.42
Insomnia	5-6 h	49	3.53	1.57	7.91
Normal sleeping	< 5 h	260	1.13	0.79	1.62
Poor sleep	< 5 h	125	2.43	1.36	4.33
Insomnia	< 5 h	64	5.12	2.22	11.79

All data adjusted for age, race, sex, BMI, diabetes, smoking status, alcohol consumption, depression, SDB, and sampling weight.
The interaction between insomnia and objective sleep duration is statistically significant, $P < 0.01$.
Compared to the common reference group, persons without insomnia/ poor sleep and slept more than 6 hours.

of insomnia (difficulty falling or staying asleep and/or early final awakening), which comprised 22% of our sample, was not associated with an increased risk for hypertension. When we introduced the criterion of objectively measured short sleep duration in the definition of insomnia, we noticed a strong and significant effect on the association of insomnia with hypertension. Chronic insomniacs who slept ≤ 5 h or 5-6 h, respectively, had a risk of hypertension 500% or 350% higher than subjects who slept > 6 h and had no sleep complaint. In contrast, insomniacs who slept ≥ 6 h did not show an increased risk for hypertension compared to the control group. Subjects with the milder form of insomnia (difficulty falling or staying asleep) had a significantly higher risk for hypertension when they slept ≤ 5 h and a nonsignificantly increased risk when they slept 5-6 hours compared to the control group. Interestingly, milder insomniacs (poor sleep) who slept ≥ 6 h showed a nonsignificantly reduced risk for hypertension. Thus, both severity/chronicity of the disorder and objective short sleep duration appear to have a strong effect on the association between insomnia and hypertension.

Our findings on the additive or synergistic effect of the insomnia complaint and objective sleep duration are consistent with previous reports that insomnia with short sleep duration is associated with hypercortisolemia,[9,10] increased catecholaminergic activity,[24] and increased autonomic activity (e.g., increased heart rate and 24-h metabolic rate and impaired heart rate variability[13-16]), all of which may lead to hypertension and other cardiovascular events. Also, a recent study showed that in adolescents, objective short sleep duration is associated with higher blood pressure.[25] Previous studies that have shown that insomnia paradoxically reduces mortality[7] might be explained by an imprecise definition of insomnia, lumping together severe and milder forms of the disorder and lack of objective measures of sleep duration.

Based on our study, about 50% of the chronic insomniacs and more than 20% of the subjects with poor sleep are at a clinically as well as statistically significant risk for hypertension and possibly other cardiovascular disorders. Because our sample is representative of the general population in the U.S., 8% to 10% of the U.S. population may suffer from a condition with significant medical complications. These findings suggest that chronic insomnia is a major public health problem and not an obsession of otherwise healthy individuals.

We have previously suggested that sleep laboratory testing is not necessary in the evaluation and diagnosis of most insomniacs with the exception when there is a clinical basis to suspect sleep apnea or restless legs/periodic limb movements.[26] The findings of this study suggest that polysomnographic measurements may provide a reliable index of the biological and medical significance and severity of chronic insomnia. Because use of the sleep laboratory to predict the medical severity of chronic

insomnia is costly and impractical, simpler methods, such as actigraphy,[27] may provide information that is equally useful to the practicing physician. Larger studies are needed to establish the reliability and validity of simple objective measures of sleep duration, for example, actigraphy versus polysomnography, in chronic insomniacs.

The field of sleep disorders medicine has attempted to define subgroups within insomnia based on etiology, e.g., primary vs. secondary, age of onset, childhood vs. adult, discrepancy between subjective and objective findings.[22] The data on the association of insomnia with hypertension, as well as previous reports on insomnia and the stress system[8-10,24] and the autonomic system,[13-16] provide the basis for a meaningful subtyping of chronic insomnia based on objective duration of sleep. One subtype is associated with physiological hyperarousal, i.e., short sleep duration, activation of the stress system, and significant medical sequelae, e.g., hypertension. The other subtype is not associated with physiological hyperarousal, i.e., normal sleep duration, normal activity of the stress system, and lack of significant medical sequelae. Other possible differences between these 2 subtypes, such as emotional and personality characteristics, daytime sleepiness and fatigue, metabolic profile, and predisposition to depression, should be the focus of future studies. These 2 subtypes may respond differentially to treatment approaches; the first subtype may respond better to medication or other biological treatment, whereas the second subtype may respond better to psychological treatment, such as cognitive/behavioral treatment. The diagnostic validity and utility of this subtyping should be tested in future studies.

Several studies have shown that self-reported sleep duration is associated with a modest increase of the risk of hypertension or other cardiovascular problems.[28-30] Our study suggests that objective short sleep duration, particularly in its extreme form ≤ 5 h is associated with a similar modest risk. However, this risk is diminished significantly in the absence of sleep complaints. Although objective sleep duration is usually shorter by 1 hour than self-reported sleep duration, the 2 variables are significantly correlated in large epidemiological samples.[31]

The association of depression with insomnia is strong and well-established.[1,2,26] Depression is often present at the onset of insomnia, whereas insomnia may precede the onset of depression. Also, depression is associated with an increased risk of hypertension and other cardiovascular problems.[32] In our study, controlling for depression did not significantly diminish the association between insomnia and hypertension. This suggests that the 2 disorders, despite the overlap, have distinct pathophysiologic mechanisms; their delineation will lead to more specific treatments.[2]

The objective sleep duration in this study was based on one night of polysomnography, which may not be representative of the subjects' habitual sleep duration. However, in our previous studies, the association between objective sleep duration and hypercortisolemia was based on a 4 consecutive night sleep laboratory protocol, which should represent better the typical sleep profile of the subjects.[8,9] The consistency of the findings on the role of objective sleep duration in predicting insomnia severity between these physiological studies with multiple night recordings and the current epidemiological study based on a single night recording increases our confidence about the replicability and generalizability of the present findings. Future studies should explore the association between insomnia, sleep duration, and hypertension using multiple night recordings. There is a possibility that blood pressure is affected more in those who tend to be nervous in a new environment. However, the recorded blood pressure was the average of 3 consecutive readings during a 5-minute period. Furthermore, insomniacs who slept < 5 h compared to those who slept > 6 h were not different in terms of anxiety or other signs of psychological distress as shown by psychometric testing (data not shown). Finally, our study is cross-sectional and does not provide causality in terms of the direction of the association. However, based on large amounts of clinical and research data, which have documented that insomnia is associated with physiologic hyperarousal,[2,8-10,13-16] the most likely direction is that insomnia leads to hypertension. It should be noted that most of the studies that established the association between SDB and hypertension were cross-sectional,[33-35] and were later confirmed by data from prospective studies.[36]

In conclusion, insomnia with short sleep duration is associated with a high risk for hypertension to a degree comparable with the other most common sleep disorder, i.e., sleep disordered breathing.[33-35] Given the high prevalence of the disorder in the general population and the widespread misconception of a disorder of the "worried well," its diagnosis and appropriate treatment should become the target of public health policy. Objective measures of sleep duration of insomnia may serve as clinically useful predictors of the medical severity of chronic insomnia and there is a need for validation of practical, easy to use, inexpensive methods to measure sleep duration outside of the sleep laboratory. Finally, insomnia with objective short sleep duration may represent a phenotype within chronic insomnia that may respond differentially to treatment.

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DISCLOSURE STATEMENT

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