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Risk Factors for Incident Chronic Insomnia: A General Population Prospective Study

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Abstract

Objective—The few population-based, prospective studies that have examined risk factors of incident insomnia were limited by small sample size, short follow-up, and lack of data on medical disorders or polysomnography. We prospectively examined the associations between demographics, behavioral factors, psychiatric and medical disorders, and polysomnography with incident chronic insomnia.

Methods—From a random, general population sample of 1741 individuals of the adult Penn State Sleep Cohort, 1395 were followed-up after 7.5 years. Only subjects without chronic insomnia at baseline (n=1246) were included in this study. Structured medical and psychiatric history, personality testing, and 8-hour polysomnography were obtained at baseline. Structured sleep history was obtained at baseline and follow-up.

Results—Incidence of chronic insomnia was 9.3%, with a higher incidence in women (12.9%) than in men (6.2%). Younger age (20–35 years), non-white ethnicity, and obesity increased the risk of chronic insomnia. Poor sleep and mental health were stronger predictors of incident chronic insomnia compared to physical health. Higher scores in MMPI-2, indicating maladaptive personality traits, and excessive use of coffee at baseline predicted incident chronic insomnia. Polysomnographic variables, such as short sleep duration or sleep apnea, did not predict incident chronic insomnia.

Conclusion—Mental health, poor sleep, and obesity, but not sleep apnea, are significant risk factors for incident chronic insomnia. Focusing on these more vulnerable groups and addressing the modifiable risk factors may help reduce the incident of chronic insomnia, a common and chronic sleep disorder associated with significant medical and psychiatric morbidity and mortality.

Keywords

chronic insomnia; incidence; physical health; mental health; general population

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Introduction

Insomnia is the most common sleep disorder, with substantial impact on the individual and the society as a whole. Some of the known consequences of insomnia include poor socio-occupational functioning, increased risk of psychiatric disorders, higher utilization of health care services, and higher risk of motor vehicle accidents [1,2]. More recent studies indicate that insomnia with objective short sleep duration is associated with significant morbidity and mortality [3–6]. Therefore, it is crucial to identify the risk factors of insomnia, which may improve prevention and treatment.

The prevalence of insomnia in the general population ranges between 7.5–30% [6–8] and is frequently associated with medical and psychiatric disorders [8–13]. The nature of the association between chronic insomnia and medical and psychiatric disorders is not established as most of these studies exploring prevalence and risk factors have been cross-sectional. Furthermore, in clinical practice, many patients with chronic insomnia report to have been “light or poor sleepers” even before developing chronic insomnia (ICSD-2); however, no large, general population epidemiological studies have examined this association.

Longitudinal studies are important in establishing the direction of the association between two conditions, i.e., mental health and chronic insomnia. The few longitudinal studies examining the risk factors of incident insomnia were limited by lack of information on medical and psychiatric disorders and certain behavioral factors (e.g., caffeine), small sample size and a relatively short duration of follow-up, or were limited to specific population groups (elderly, men) [14–18]. Furthermore, none of these studies included objective measures of sleep (i.e., polysomnography) at baseline.

In this study we examined the association of demographics, behavioral factors, psychiatric and medical disorders, MMPI-2 personality traits, and polysomnography with subsequent onset of insomnia 7.5 years after the initial evaluation in a large general population sample of men and women.

Methods

Population

The data presented here were collected as part of a population-based study to establish the age distribution of sleep disordered breathing, which used a two-phase protocol in order to recruit participants from various age groups [19,20]. The study was approved by the University’s Institutional Review Board. 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 two counties of Central Pennsylvania (Dauphin and Lebanon) using the Mitofsky–Waksberg two-stage random digit dialing procedure. A within-household selection procedure described by Kish was used to select the specific man or woman to be interviewed [21]. 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 randomly from those subjects previously interviewed by telephone were studied in our sleep laboratory. After giving a complete description of the study to the subjects, written informed consent was obtained. Data were collected between January 1990 and March 1999. 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, and

prevalence of sleep disorders. There were no significant differences between these two groups on any of these variables.

Of the 1741 subjects who completed the comprehensive sleep evaluation, including the sleep laboratory recording, 1395 subjects were followed up after an average duration of 7.5 years (mean duration of 4.5 years for women and 10.5 years for men) by one of the investigators (S.C.) via telephone interview. Data were collected between June 2000 and April 2004. In the Penn State Cohort Study, men were recruited first and women five years later. This explains the five year difference in the follow-up period between men and women. The overall response rate was 79.7%. However, among the 1741 subjects, 215 were deceased, resulting in a response rate of 90.9% of those alive. After complete description of the follow-up study to the subjects, verbal informed consent was obtained.

Key Measurements

At baseline a structured history of sleep disorders/complaints, common medical and psychiatric disorders, cigarette smoking, and caffeine and alcohol use was obtained. All subjects were also evaluated for one night in the sleep laboratory in sound-attenuated and light and temperature controlled rooms. During this evaluation, each subject was continuously monitored for eight hours using 16-channel polygraphs including electroencephalogram, electrooculogram, and electromyogram. Bedtimes were adjusted to conform to subjects' usual bedtimes and subjects were recorded between 22:00–23:00 and 06:00–07:00. In this random general sample of Central Pennsylvania, the vast majority of individuals went to bed between 10 PM and 11 PM, whereas only a small minority went to sleep outside of this time window and none for more than 1 hour, thereby making a 1-hour maximum adjustment. The sleep records were scored independently according to standardized criteria [22]. Respiration was monitored throughout the night by use of thermocouples at the nose and mouth and thoracic strain gauges. All-night hemoglobin oxygen saturation (SpO₂) was obtained with a finger oximeter. For the purpose of this study, sleep apnea was defined as an apnea or hypopnea index of five or greater (AHI $\geq$ 5).

As part of the protocol at baseline we also assessed for the presence of all sleep disorders, which was based on a standardized questionnaire completed by the subjects on the evening of their sleep laboratory visit. The characteristics and content of this questionnaire have been extensively presented elsewhere [3,7]. This questionnaire consists of 53 questions (7 demographic, 20 sleep related, and 26 general health questions). In addition, women responded to eight questions related to menstrual history, menopause, and hormone therapy. Sleep related questions were qualified in terms of severity on a scale of 0–3 (0 = none, 1 = mild, 2 = moderate, 3 = severe) and duration. Health problems 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 three levels of severity. First, *chronic insomnia* was defined by a complaint of insomnia (i.e., a positive response to the question “Do you feel you have insomnia?”) with a duration of at least one year. Second, *poor sleep* was defined as a positive response to one or more of moderate to severe complaints (based on the 4-point likert scale with none, mild, moderate and severe options mentioned above) of difficulty falling asleep (“Do you have difficulty falling asleep?”), difficulty staying asleep (“Do you have difficulty staying asleep?”), early morning awakening (“Do you wake up in the morning earlier than desired?”), or unrefreshing sleep (“Do you still feel groggy and unrefreshed after morning awakening?”). Finally, *normal sleeping* was defined as the absence of either of these two categories. In order to create three mutually exclusive categories, none in the poor sleep group reported having chronic insomnia and none in the normal sleeping group reported either chronic insomnia or poor sleep. Polysomnographic variables were not used to define either poor sleep or chronic insomnia.

The presence of medical and psychiatric disorders at baseline was determined by obtaining a structured history of current or past diagnosis or treatment of all major chronic disorders. Alcohol use disorder was ascertained by patient report of current or past diagnosed alcohol use disorder. Cigarette smoking was assessed by enquiring if the subjects smoked or not and if they were smoking, how many cigarettes did they smoke per day. Similarly, caffeine and alcohol consumption at baseline was assessed by enquiring about the number of cups of caffeinated beverages per day or alcoholic drinks per day, respectively.

To assess the personality profile of the subjects, the MMPI-2 was administered at baseline following the standardized rules and scored accordingly [23]. The MMPI-2 has 10 clinical scales that are universally scored when the MMPI-2 is administered. These clinical scales are numbered zero through nine and include: 1-Hypochondriasis, 2-Depression, 3-Hysteria, 4-Psychopathic Deviate, 5- Masculinity-Femininity, 6-Paranoia, 7-Psychasthenia, 8-Schizophrenia, 9-Hypomania, and 0-Social Introversion. We also examined the commonly used three research scales Depression, Repression, and Ego Strength, which are primarily derived from the main clinical scales. The Masculinity-Femininity scale was not used because the sample was comprised of both men and women.

Follow-up measures taken through telephone interview by one of the investigators (S.C.) included an identical standardized questionnaire that subjects completed at baseline during their sleep laboratory visit. Sleep-related questions were also used to establish the presence of sleep difficulty at follow-up based on three levels of severity (i.e., *normal sleeping*, *poor sleep*, and *chronic insomnia*), as defined above.

Sample

Of the 1395 subjects who were followed up, 149 subjects had *chronic insomnia* at baseline. Because the focus of the present study was the incidence of insomnia, those individuals with *chronic insomnia* at baseline were excluded from the analysis. The remaining 1246 subjects selected for the present study were further classified into two groups according to their follow-up insomnia status: *no chronic insomnia* (individuals without chronic insomnia both at baseline and at follow-up; $n = 1113$), and *incident chronic insomnia* (individuals without chronic insomnia at baseline and with chronic insomnia at follow-up; $n = 133$). Figure 1 shows the participant flow in the study.

Statistical Analysis

The design of this study included oversampling of those at higher risk for sleep disordered breathing and women with markedly higher levels of BMI to increase the precision of the risk estimates. Because of this sampling strategy, numeric weights were developed for the analysis so that estimates could be obtained for the original target population of men and women in the 2-county study area [19,20]. A comprehensive presentation of these numeric weights has been reported elsewhere [3,4,8,19,20]. Specifically, three 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 three age groups in order to oversample the middle age group. The final weight for the men was computed to account for the oversampling of subjects for the sleep laboratory study (Phase II); those with larger counts of the four possible risk factors, i.e., snoring, daytime sleepiness, obesity, and hypertension, had substantially higher probability of being selected. For the women, the only weight required was to account for the oversampling of subjects for the sleep laboratory study. To eliminate any possible sample bias due to oversampling different strata of the target population, we calculated 32 unique

weights for the women and 16 unique weights for the men corresponding to all possible combinations of the five risk factors for the women (menopause was the fifth risk factor) and four for the men. Any individual weight that had too small of a cell size was combined with adjacent cells so that less than 10% of the cells had a sample < 25 and no cell had a size less than 10. Finally, we used the BMI and race distributions by age decade from the NHANES III laboratory data as the standard (24) to adjust both the men and women in terms of BMI and race to be representative of the national population. All statistical analyses were adjusted for the sampling weight to make appropriate inference to the target population.

For descriptive statistics, we obtained means (standard deviations) and proportions as appropriate for the entire sample, as well as stratified by each group. Multivariate logistic regression was used to examine the relative association between various demographic, behavioral, psychiatric, and medical disorders among the groups. To explore the relative association of mental vs. physical health we entered in the multivariate models two composite variables of physical (i.e., allergy/asthma, anemia, kidney/bladder, and migraine) and mental (i.e., depression and alcohol use disorder) health problems. A one-way between groups multivariate analysis of covariance (MANCOVA) was performed to investigate group differences in mean MMPI-2 scores on various clinical and research scales. Unless otherwise noted, $p=0.05$ was used to determine statistical significance. All statistical analyses were conducted using SPSS statistical package version 17.0.

Results

The demographic and behavioral characteristics of the overall study sample and stratified by incident chronic insomnia status are presented in Table 1. One hundred and thirty three subjects developed insomnia after an average of 7.5 years of follow-up, reflecting a sampling weight adjusted incidence of 9.3%. Women, young adults (20–35 years), non-whites, and obese individuals had a significantly higher risk of developing insomnia. Moreover, individuals who consumed on average >3 cups of coffee had the highest incidence of chronic insomnia. Interestingly, individuals who drank on average one alcoholic drink per day had the lowest risk of chronic insomnia in comparison to individuals who did not drink alcohol or who drank ≥ 2 drinks per day.

As shown in Table 2, individuals with a history of poor sleep were at significantly higher risk of developing chronic insomnia. In contrast, PSG parameters, including objective short sleep duration (Table 2) or sleep apnea (Table 3), did not predict the development of chronic insomnia.

Moreover, incident chronic insomnia was significantly higher in subjects with physical and mental health problems, such as alcohol use disorder, depression, migraine, kidney/bladder disorders, and, to a lower degree, allergy/asthma and anemia (see Table 3).

We employed four sets of multivariate logistic regression models to examine the association of each risk factor after progressively adjusting for those that were significant in the above mentioned univariate analyses (see Table 4). Female gender, younger age, non-white ethnicity, and consuming ≥ 3 cups of coffee were all significant risk factors for incident chronic insomnia. Obesity, however, showed a marginal significant association ($OR=1.47$; $CI=.96-2.26$) with incident chronic insomnia after controlling for sociodemographic and behavioral factors, (Table 4, model 1). Physical health problems were significantly associated with incident chronic insomnia (Table 4, model 2); however, after adjusting for mental health problems, which were strongly associated with incident chronic insomnia, physical health did not remain as a significant predictor (Table 4, model 3). Importantly,

poor sleep remained as an independent risk factor for incident chronic insomnia after adjusting for all other demographic, behavioral, physical health, and mental health risk factors.

Finally, in order to test whether certain personality characteristics increase the risk for new onset chronic insomnia, we further examined in a subsample of subjects that completed the MMPI-2 at baseline ($n = 980$) whether differences existed in terms of the personality profile of those individuals who developed chronic insomnia and those who did not. As shown in table 5, incident chronic insomniacs presented higher MMPI-2 scores in depression, social introversion, and repression, and lower scores in ego strength at baseline.

Discussion

Our study shows that the complaint of poor sleep and the presence of mental health problems are stronger predictors of incident chronic insomnia compared to physical health problems. Furthermore, neither objective short sleep duration nor sleep apnea significantly predicted incident chronic insomnia.

The incidence of insomnia was similar to the rates in previous studies [7,14,16]. Previous studies [16,17] followed subjects for one year and neither assessed the presence of medical disorders, nor controlled for sleep apnea and certain demographic (BMI) and behavioral factors (e.g., caffeine). Female gender and non-white race increased the risk of incident insomnia as reported in a previous study [25]. We found that the risk of insomnia was higher in younger subjects aged 20–35 years than in older subjects aged 65 years or more. The younger subjects may be at increased risk for experiencing significant life stressors (i.e., marriage, birth of children), vulnerability to stress [26], and physiological sleep changes than older subjects, which may in turn increase their risk of insomnia. Since we excluded individuals with insomnia at baseline, the age difference in incidence of insomnia could also be due to the “age survival effect” in that older baseline insomnia-free individuals are healthier and less vulnerable to incident insomnia than their younger counterparts.

In the present multivariate analysis, mental health problems (i.e., depression and alcohol use disorder) were strong predictors of incident chronic insomnia. These findings are partially consistent with previous longitudinal studies [16,17], which found increased risk of incident insomnia in subjects with depression and anxiety at baseline. Also, physical health problems (e.g., kidney/bladder) significantly increased the risk of incident chronic insomnia. These results are consistent with previous cross-sectional reports of increased prevalence of insomnia and sleep disturbances in clinical samples of chronic kidney disease and urinary problems [13,27]. An additional new finding of our study is that mental health was a stronger predictor of incident chronic insomnia compared to physical health. Finally, incident chronic insomniacs had a premorbid psychological profile of higher neuroticism and excessive rumination with a tendency to suppress negative emotional content (internalization) and a decreased ability to cope, as revealed by higher scores on MMPI-2 scales of depression, social introversion, and repression, and lower scores on the ego strength scale. These findings are consistent with previous cross-sectional data [4,28–30] and confirm that this personality profile may be a trait dimension that precedes the onset of chronic insomnia.

Most importantly, a history of subjective poor sleep increased the risk of new onset chronic insomnia. The presence of subjective poor sleep at baseline doubled the odds of developing chronic insomnia compared with individuals without poor sleep independent of demographic and behavioral factors, co-morbid medical and psychiatric disorders, and sleep apnea at baseline. Polysomnographic variables, such as sleep duration, sleep latency, and

wake time after sleep onset, did not predict incident chronic insomnia. Our results are consistent with clinical observations of chronic insomniacs who typically report “poor” or “light” sleep before they develop clear-cut chronic insomnia [16,31]. This suggests that the complaint of subjective poor sleep in young age may be a marker of developing chronic insomnia later in life when physiological sleep mechanisms become relatively impaired. These results emphasize the importance of assessing for even mild sleep complaints during routine physical examinations or during any contact with a health care provider to prevent the development of chronic insomnia later in life.

A novel finding of our study is that obesity is a risk factor for new-onset chronic insomnia. We have previously reported that sleep complaints are twice as frequent in obese individuals compared to the non-obese [32]. Many studies, both cross-sectional and longitudinal, have reported that subjective short sleep duration is associated with obesity. The nature of this association is not clear since subjective sleep duration is a marker of psychosocial stress [32]. Our longitudinal study suggests that obesity increases significantly the risk of new-onset chronic insomnia. Future research should explore whether chronic insomnia or poor sleep are risk factors for obesity.

Consistent with our previous cross-sectional study [7], sleep apnea was not significantly associated with incident chronic insomnia in the present study. Although some investigators have suggested that sleep apnea may be a cause of chronic insomnia based on cross-sectional studies on clinical samples [33], our prospective study indicates that sleep apnea does not increase the risk of chronic insomnia. Furthermore, objective short sleep duration was not a risk factor for new onset insomnia. This result, combined with our previous findings [3–6], suggests that objective short sleep duration is an index of severity when combined with chronic insomnia and not a risk factor on its own.

High caffeine intake was associated with the highest risk for insomnia, whereas subjects who reported moderate caffeine consumption (1–2 cups of coffee/day) had the lowest risk of incident insomnia. The risk of developing insomnia was intermediate with no caffeine use. Although the latter is counterintuitive, the most likely explanation is that those who avoid caffeine may be more sensitive to caffeine effects, and they have learned to avoid caffeine use. That moderate use of caffeine is associated with less risk of insomnia may indicate that in normal sleepers chronic moderate use of this substance does not affect sleep. We did not find a significant relationship between moderate alcohol consumption and insomnia risk. These results are consistent with some [10,34], but not all [25], epidemiological studies of insomnia. An interesting question rises on how we reconcile the finding that alcohol use disorder is a risk factor for incident insomnia whereas there is no association between moderate use of alcohol and incident insomnia. The most likely explanation of these seemingly discrepant results is that people with “light” sleep tend to avoid the use of alcoholic drinks to protect themselves from the well-known disturbing effects of alcohol on sleep. In contrast, people with “sound” sleep may use moderate amounts of alcohol and not experience the physiological effects of alcohol on their sleep. Light sleepers may therefore learn to avoid these substances while sound sleepers use them in a moderate way because they do not disturb their sleep. Another, not mutually exclusive explanation, is that moderate alcohol consumption may have a beneficial effect on mood [35] and cardiovascular health [36], thus protecting individuals from developing insomnia.

There are some limitations that should be considered in interpreting the results of this study. Even though we used a stringent duration criteria of one year to define incident chronic insomnia, the diagnosis was based on a single question and not on a complete diagnostic interview, which is common in large epidemiological studies. Furthermore, objective sleep measures in this study were based on one night of PSG, which may not be representative of

the subjects' typical objective sleep duration and may be affected by the so-called "first night effect." However, in large epidemiologic studies the average objective sleep duration is about six hours, which is independent of whether sleep is recorded at home with polysomnography (Sleep Heart Health Study; 37), for three consecutive nights with actigraphy (CARDIA study, 38), or in the sleep laboratory (Penn State Cohort, 7). Thus, the consistency among these three large epidemiological studies in terms of objective sleep duration supports the validity, replicability, and generalizability of our findings. Additionally, it is likely that work schedules, particularly shift-work, may have an influence on future development of insomnia. Unfortunately we did not obtain information on work schedules. Finally, the diagnosis of depression and other disorders was based on a single question (physician-based diagnosis) and not on a structured diagnostic interview, which is common in large epidemiological studies.

In conclusion, our large, prospective study, which followed-up individuals for an average of 7.5 years, showed that mental health, poor sleep, and obesity, but not sleep apnea, are significant predictors of incident chronic insomnia. Furthermore, our study demonstrated that younger individuals are more likely to develop chronic insomnia compared to older individuals. Our findings suggest that focusing on young, obese individuals and those that complain of poor sleep and poor mental health may help to reduce the rate of new-onset chronic insomnia.

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Abbreviations

AHI	apnea / hypopnea index
BMI	body mass index
MANCOVA	multivariate analysis of covariance
MMPI-2	Minnesota Multiphasic Personality Inventory
PSG	polysomnography

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Figure 1.
Participants' flow in the study

Table 1

Baseline demographic and behavioral characteristics of study sample

	All (n = 1246)	No chronic insomnia (n=1113)	Incident Chronic Insomnia (n = 133)	<i>P</i>
Gender				
Females, %	47.6	45.8	65.5	<.001
BMI	27.5 (5.1)	27.5 (4.9)	27.9 (7.00)	.40
Obesity, %	23.9	23.1	32.0	.03
Age	49.0 (13.1)	49.4 (13.0)	45.5 (13.6)	.002
20–35, %	13.9	12.6	26.2	<.001
>35– <65, %	72.3	73.1	64.7	
>65, %	13.8	14.3	9.1	
Race				
Non-white, %	9.7	8.4	22.9	<.001
Caffeine				
None, %	32.4	32.9	30.4	.004
1–2 cups/day, %	31.5	32.8	21.2	
>3 cups/day, %	35.5	34.4	48.4	
Cigarettes				
None, %	78.7	78.6	80.6	.65
1–20, %	16.0	16.0	16.0	
>20, %	5.3	5.4	3.5	
Alcohol				
None, %	72.1	71.6	82.4	.04
1 drink/day, %	10.6	11.2	5.8	
>2 drinks/day, %	16.6	17.3	11.9	

All data are adjusted for sampling weight. Where appropriate the standard deviation is presented in parenthesis. BMI = Body Mass Index. Obesity = BMI ≥ 30.

Table 2

Baseline subjective and objective (polysomnographic) sleep characteristics of study sample

		No Chronic Insomnia (n = 1113)	Incident Chronic Insomnia (n =133)	P
Subjective sleep				
DFA	Yes,%	19.1	39.2	<.001
	No,%	80.9	60.8	
DSA	Yes,%	18.0	30.0	<.001
	No,%	82.0	70.0	
EMA	Yes,%	20.8	30.0	<.001
	No,%	79.2	70.0	
NRS	Yes,%	8.7	26.9	<.001
	No,%	91.3	73.1	
Poor sleep	Yes,%	20.4	41.7	<.001
	No,%	79.6	58.3	
Objective sleep				
Sleep latency		32.27 (0.92)	32.40 (2.91)	.97
Wake after sleep onset		91.06 (1.47)	90.26 (4.63)	.87
Total wake time		123.33 (1.90)	122.66 (5.99)	.92
Number of awakenings		11.46 (0.17)	10.70 (0.53)	.17
Percent of Stage 1		11.32 (0.22)	10.77 (0.71)	.45
Percent of Stage 2		68.83 (0.27)	70.11 (0.85)	.15
Percent of Stage 3		2.77 (0.13)	2.29 (0.39)	.24
Percent of Stage 4		0.30 (0.04)	0.31 (0.12)	.93
Percent of SWS		3.07 (0.14)	2.60 (0.44)	.31
Percent of REM		16.78 (0.20)	16.53 (0.63)	.70
Total Sleep Time		352.21 (1.94)	354.93 (6.10)	.67
	≥ 6 hr, %	56.1	55.4	.87
	< 6 hr, %	43.9	44.6	
Percent of Sleep Time		74.1 (0.40)	74.3 (1.26)	.85
Apnea/hypopnea index		2.68 (0.23)	3.31 (0.72)	.40

DFA = difficulty falling asleep. DSA = difficulty staying asleep. EMA= early morning awakening. NRS = non-restorative sleep. Poor sleep = a positive response to one or more of DFA, DSA, EMA, and/or NRS (see Methods). Objective sleep data are adjusted for age, gender, race, BMI, and sampling weight. Where appropriate the standard error of the mean (SEM) is presented in parenthesis.

Table 3

Univariate association between mental and physical health disorders at baseline and incident chronic insomnia after 7.5 years of follow up.

		Incident Chronic Insomnia (n = 133)		
		OR	95%CI	P
Mental health problems, %	Yes	18.1		
	No	7.9	2.59 1.68–4.00	<.001
Depression, %	Yes	15.6		
	No	8.4	2.02 1.27–3.21	.003
Alcohol disorder, %	Yes	32.3		
	No	9.0	4.83 1.89–12.37	<.001
Physical health problems, %	Yes	12.1		
	No	7.6	1.67 1.15–2.44	.007
Allergy/Asthma, %	Yes	11.9		
	No	8.5	1.46 0.97–2.18	.07
Anemia, %	Yes	14.7		
	No	8.9	1.77 0.99–3.17	.05
Colitis, %	Yes	12.6		
	No	9.3	1.41 0.48–4.14	.53
Diabetes, %	Yes	8.6		
	No	9.4	0.91 0.38–2.17	.83
Epilepsy, %	Yes	7.0		
	No	9.4	0.73 0.10–5.48	.76
Heart disorder, %	Yes	8.3		
	No	9.4	0.87 0.43–1.75	.69
Hypertension, %	Yes	11.8		
	No	8.8	1.39 0.88–2.19	.15
Kidney/Bladder, %	Yes	15.2		
	No	8.8	1.85 1.04–3.29	.03
Migraine, %	Yes	15.2		
	No	8.6	1.91 1.17–3.13	.009
Rheumatism, %	Yes	11.9		
	No	9.2	1.34 0.68–2.63	.40

		Incident Chronic Insomnia (n = 133)		OR	95%CI	P
Thyroid, %	Yes	14.8		1.74	0.81–3.72	.15
	No	9.1				
Ulcer, %	Yes	4.5		0.44	0.13–1.48	.17
	No	9.6				
Sleep apnea, %	Yes	7.4		0.75	0.41–1.38	.35
	No	9.6				

All data adjusted for sampling weight; OR = odds ratio; 95%CI = 95% confidence interval. Mental health problems = composite variable of depression and alcohol use disorder. Physical health problems = composite variable of those medical disorders in which significant differences were found between groups (i.e., allergy/asthma, anemia, kidney/bladder, and migraine). Sleep apnea = apnea/hypopnea index ≥ 5 .

Table 4

Summary of multivariate regression modeling of demographic, behavioral, physical disorders, mental disorders, and poor sleep at baseline as predictors of incident chronic insomnia

	Model 1	Model 2	Model 3	Model 4
	OR (95%CI)	OR (95%CI)	OR (95%CI)	OR (95%CI)
Female gender	2.05 (1.34–3.13) **	2.01 (1.32–3.08) **	1.87 (1.22–2.88) **	1.79 (1.15–2.77) **
Obesity	1.47 (0.96–2.26)	1.44 (0.93–2.22)	1.39 (0.90–2.15)	1.24 (0.80–1.93)
Age	0.98 (0.96–0.99) **	0.98 (0.96–0.99) **	0.98 (0.96–0.99) **	0.98 (0.96–0.99) **
Non-white race	2.31 (1.21–4.44) *	2.47 (1.28–4.77) **	2.56 (1.33–5.01) **	2.84 (1.45–5.57) **
Caffeine^a				
1–2/d	0.70 (0.41–1.20)	0.72 (0.42–1.23)	0.73 (0.43–1.26)	0.64 (0.37–1.12)
≥ 3/d	2.02 (1.26–3.23) **	2.11 (1.31–3.39) **	1.98 (1.23–3.20) **	1.88 (1.16–3.04) *
Alcohol^a				
1/d	0.50 (0.22–1.14)	0.53 (0.23–1.20)	0.58 (0.25–1.31)	0.60 (0.26–1.38)
≥ 2/d	0.72 (0.38–1.34)	0.72 (0.38–1.35)	0.77 (0.41–1.44)	0.78 (0.41–1.48)
Physical health				
		1.56 (1.06–2.30)	1.42 (0.95–2.12)	1.40 (0.93–2.09)
Mental health				
			2.00 (1.24–3.24) **	1.65 (1.01–2.70) *
Poor sleep				2.21 (1.44–3.40) **

Model 1. Factors included in the model: age, gender, obesity, race, cigarettes, alcohol, and caffeine use.

Model 2. All factors in model 1 and physical health problems (i.e., allergy/asthma, anemia, kidney/bladder, and migraine) and sleep apnea.

Model 3. All factors in model 2 and mental health problems (i.e., depression and alcohol use disorder).

Model 4. All factors in model 3 and poor sleep.

* p < .05;

** p < .01

^aReference group for alcohol (drinks/day) and caffeine (cups/day) = None / day

All data are adjusted for sampling weight. OR = odds ratio; 95%CI = 95% confidence interval

Table 5

MMPI-2 mean scores in subjects who developed chronic insomnia compared to subjects who did not develop chronic insomnia at follow-up.

	No Chronic Insomnia (n=876)	Incident Chronic Insomnia (n=104)	<i>P</i>
Clinical Scales			
1-Hypochondriasis	54.41 (9.62)	56.14 (10.92)	.12
2-Depression	49.82 (10.86)	53.63 (12.32)	.003
3-Hysteria	53.25 (10.06)	52.36 (11.43)	.45
4-Psychopathic deviate	51.01 (10.12)	49.71 (11.48)	.27
6-Paranoia	50.04 (10.12)	50.64 (11.47)	.61
7-Psychasthenia	49.76 (10.03)	50.12 (11.38)	.77
8-Schizophrenia	48.08 (9.50)	48.08 (10.78)	.99
9-Hypomania	47.87 (9.56)	48.61 (10.84)	.51
0-Social Introversion	48.91 (9.68)	51.23 (10.97)	.039
Research Scales			
Depression	46.70 (9.59)	49.24 (10.87)	.023
Repression	50.09 (9.09)	52.01 (10.32)	.07
Ego Strength	48.97 (9.12)	46.17 (10.34)	.008

All data adjusted for age, gender, race, obesity, and sampling weight. The standard deviation is presented in parenthesis (SD).