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Lower blood pressure and smaller pulse pressure in sleeping pill users

A large-scale cross-sectional analysis

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Abstract

This study aimed to investigate the association between sleeping pill use and hypertension or blood pressure (BP) via a cross-sectional analysis.

A total of 11,225 subjects (5875 men and 5350 women) underwent health examinations. We compared the proportion of sleeping pill users among hypertension (n = 5099) and normotensive (n = 6126) participants. We analyzed participants with no intake of antihypertensive medication (n = 7788), comparing the proportions with high systolic BP (SBP) ≥140, high diastolic BP (DBP) ≥90, and high pulse pressure (PP) ≥50 mm Hg across 3 subgroups. These groups were classified according to the sleeping pill use [nonuse group (n = 6869); low-frequency-use group, defined as taking sleeping pills ≤2 days per week (n = 344); and high-frequency-use group, defined as taking sleeping pills ≥3 days per week (n = 575)].

In the multivariable-adjusted model, odds of sleeping pill use (odds ratio (OR), 1.14; $P < .05$) was significantly higher in the hypertensive group compared with the normotensive group. In participants with no intake of antihypertensive medication, odds of high SBP (OR, 0.65; $P < .0005$), high DBP (OR, 0.58; $P < .005$), and high PP (OR, 0.77; $P < .01$) were significantly lower in the high-frequency-use group compared with the nonuse group. Odds of high DBP (OR, 0.59; $P < .05$) was significantly lower in the low-frequency-use group.

Sleeping pills were more frequently required in hypertensive participants than in the normotensive ones. Sleeping pill use may decrease BP and assist in the treatment of high BP in patients with sleep disturbances.

Abbreviations: BMI = body mass index, BP = blood pressure, DBP = diastolic BP, OR = odds ratio, OSA = obstructive sleep apnea, PP = pulse pressure, PSQI = Pittsburgh Sleep Quality Index, SBP = systolic BP, SNS = sympathetic nervous system, SWS = slow-wave sleep.

Keywords: hypertension, pulse pressure, sleep, sleeping pill

1. Introduction

Several studies have reported that sleep disorders and insomnia are associated with an increased risk of hypertension[1,2] and that patients with hypertension often suffer from insomnia.[3] This evidence suggests that sleeping pills are frequently required for patients with hypertension. Sleeping pills are commonly used for the treatment of sleep disorders and insomnia, and can effectively reduce sleep-related complaints, elongate subjective sleep duration, and improve sleep quality.[4] Recently, several studies have reported that inappropriate sleep duration was associated with prevalent and incident hypertension.[5–7] A recent large-scale cross-sectional study revealed a significant association between sleep quality and the presence of hypertension.[8] Fung et al[9] reported, in a cohort study, that the percentage of slow-wave sleep (SWS) was inversely associated with incident hypertension, suggesting that poor sleep quality is involved in development of hypertension. Furthermore, the anxiety that is often accompanied by sleep disorders is associated with increased blood pressure (BP).[10,11] This evidence suggests that treatment of sleep disorders and insomnia using sleeping pills may have beneficial effects on BP. In addition, several experimental studies have suggested that certain sleeping pills may decrease BP or sympathetic nervous system (SNS) activity[12–17]; however, no large-scale studies have investigated the association between hypertension and sleeping pill use, and the impact of sleeping pills on BP.

In the present study, we investigated the association between hypertension and sleeping pill use, considering age-related differences and presence of comorbidities, and its effects on BP and pulse pressure (PP) in a large sample of participants who underwent annual health examinations.

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2. Methods

2.1. Study design and participants

In November 2014, we commenced the Hiroshima Sleep and Healthcare study (HIRSH study),[18,19] a cross-sectional and cohort study that addressed the association between sleep habits and lifestyle-related diseases. Potential participants were recruited after undergoing health examinations at the Health Management and Promotion Center of the Hiroshima Atomic Bomb Casualty Council or cooperating facilities, on the basis of the following criteria: subjects were aged 18 years or older, were able to walk unassisted, did not engage in shift work, and had no history of treatment for psychological disorders (excluding insomnia treated by a general physician) or neurological diseases. All participants were questioned about their regular medications and medical histories, including treatment for hypertension, diabetes mellitus, dyslipidemia, and cardiovascular diseases, psychiatric diseases, or neurological diseases and their drinking and smoking habits. In the present study, we performed cross-sectional analyses of participants recruited between November 2014 and September 2015, using baseline data from the HIRSH study. Of these, 264 participants were excluded due to incomplete answers on the sleep questionnaire, 293 for having no BP measurements, 26 for undergoing treatment for cancer, and 1739 for undergoing treatment for, or having a history of cardiovascular diseases. A total of 11,225 subjects [5875 men and 5350 women with a mean age ± standard deviation (SD) of 67.0 ± 15.1 years and a mean body mass index (BMI) ± SD of 22.9 ± 3.3 kg/m^2], each with a complete dataset, were included in our analyses. Of these, 5099 participants (45%) had hypertension (defined as taking antihypertensive medication and/or having systolic BP (SBP) ≥140 and/or diastolic BP (DBP) ≥90 mm Hg)[20]; 1040 (9%) had diabetes (defined as taking antidiabetic medication); 2212 (20%) had dyslipidemia (defined as taking antihyperlipidemic medication); 1376 (12%) were current smokers (defined as having a current smoking habit regardless of the number of cigarettes smoked per day); and 3410 (30%) were habitual drinkers (defined as drinking alcohol for ≥5 days per week regardless of the amount consumed). Informed consent was obtained from all participants. Ethical approval for this study was obtained from the Hiroshima Atomic Bomb Casualty Council Committee on the Ethics of Human Research.

2.2. Measures

Sleep habits were evaluated using the Japanese version of the Pittsburgh Sleep Quality Index (PSQI).[21,22] The PSQI is a 19-item, self-report questionnaire used to assess subjective sleep quality and degree of sleep difficulties over the past month. It contains a 7-component scale comprising the following factors: sleep quality (C1); sleep latency (C2); sleep duration (C3); sleep efficiency (C4); sleep disturbance (C5); sleeping pill use (C6); and daytime dysfunction (C7). Scores on the seven components are then summed to yield a global PSQI score with a range of 0 to 21; higher scores indicate poorer sleep quality. Based on the questionnaire, we assessed the sleeping pill use using the following question: "During the past month, how often have you taken medicine to help you sleep?" Participants selected their response from the following options: not during the past month; less than once a week; once or twice a week; or 3 or more times a week. Using a digital, automatic BP-measuring instrument (Terumo Co., Tokyo, Japan or Omron Healthcare Co., Ltd, Kyoto, Japan), BP was measured in a seated position on a chair with a support for the back, and a support for the arm at heart level after the participant had rested for more than 5 minutes when the participants underwent health examinations. In participants with no intake of antihypertensive medication, high SBP and high DBP were defined as SBP ≥140 mm Hg and DBP ≥90 mm Hg, respectively.[20] High PP was defined as PP ≥50 mm Hg (PP was calculated as SBP minus DBP).[23]

2.3. Classification of participants to investigate the association between hypertension and sleeping pill use

Sleeping pill use was defined as intake of sleeping pills at least once during the past month. We divided the participants of this category (n = 1686) into 2 groups to investigate the age-related differences in the relationship between hypertension and sleeping pill use: younger group (age <65 years, n = 3511) and elderly group (age ≥65 years, n = 7714).

2.4. Classification of participants to investigate the impact of sleeping pill use on blood pressure

We divided the participants with on intake of antihypertensive medication (n = 7788) into 4 groups according to age: <65 years (n = 3125); ≥65 to <75 years (n = 2567); ≥75 to <85 years (n = 1659); and ≥85 years (n = 437) to investigate the association between the sleeping pill use and BP. Furthermore, we divided the participants into 3 groups according to the sleeping pill use: participants who did not use sleeping pills during the past month (nonuse group); participants who took sleeping pills ≤2 days per week (low-frequency-use group); and participants who took sleeping pills ≥3 days per week (high-frequency-use group).

2.5. Statistical analyses

Continuous variables are expressed as mean ± SD. Normality of the continuous variables was examined with the Kolmogorov–Smirnov test. Then, differences between the 2 groups were compared using the Wilcoxon rank-sum test, differences among 3 groups were analyzed by the Kruskal–Wallis test, and Dunn post hoc test was used for multiple comparison testing. Categorical variables were summarized as percentages and were analyzed using the Chi-squared test. The relationship between the sleeping pill use and hypertension was assessed with univariate and multivariate logistic regression analysis. The following 2 multivariate models were used: model 1, adjusted for age, gender, BMI, current smoker (yes or no) and habitual drinker (yes or no); model 2, additionally included the presence of diabetes (yes or no) and dyslipidemia (yes or no). The relationship between the sleeping pill use and high SBP, high DBP, and high PP was assessed with 2 models: the first adjusted for age; the second additionally included gender, BMI, current smoker (yes or no), habitual drinker (yes or no), and presence of diabetes (yes or no) and dyslipidemia (yes or no). *P*-values <.05 were considered statistically significant. All statistical analyses were performed, using the JMP 10 statistical software (SAS Institute, Inc., Cary, NC).

3. Results

3.1. Association of hypertension with sleeping pill use

The characteristics of normotensive and hypertensive groups are presented in Table 1. Of the 11,225 participants, 6126 (55%)

Table 1

Characteristics of hypertensive and normotensive study participants.

	All	NT	HT	*P*
N	11225	6126	5099	
Mean age, y	67.0±15.1	61.9±16.7	73.1±10.7	<.0001
Female, n (%)	5350 (48)	2949 (48)	2401 (48)	.27
BMI, kg/m^2	22.9±3.3	22.3±3.0	23.7±3.4	<.0001
Diabetes, n (%)	1040 (9)	387 (6)	653 (13)	<.0001
Dyslipidemia, n (%)	2212 (20)	823 (13)	1389 (27)	<.0001
Current smoker, n (%)	1376 (12)	866 (14)	510 (10)	<.0001
Habitual drinker, n (%)	3410 (30)	1601 (26)	1809 (35)	<.0001
Use of sleeping pills, n (%)	1686 (15)	735 (12)	951 (19)	<.0001

Data are expressed as mean±standard deviation or frequency and percentage.
BMI=body mass index, HT=hypertensive, NT=normotensive.

were normotensive and 5099 (45%) were hypertensive. Mean age and BMI were significantly higher in the hypertensive group than in the normotensive group. The proportion of females was similar in the 2 groups, whereas that of sleeping pill users was significantly higher in the hypertensive group than in the normotensive group (19% vs 12%, $P<.0001$). Figure 1 shows that the proportion of participants using sleeping pills was higher in both the younger and elderly hypertensive groups than in their normotensive counterparts (6.9% vs 4.0%, 20.6% vs 18.7%, respectively). The univariate and multivariate adjusted odd ratios (ORs) for sleeping pill use are presented in Table 2. In the full adjusted model (model 2), odds of sleeping pill use (OR, 1.14; 95% CI, 1.02–1.28; $P<.05$) were significantly higher in the hypertensive group compared with the normotensive group. When participants were divided according to the age, in model 1, the odds of sleeping pill use were significantly higher in both the younger and elderly hypertensive groups than in their normotensive counterparts; however, in the full adjusted model, the odds of sleeping pill use (OR, 1.49; 95% CI, 1.00–2.18; $P<.05$) were significantly higher in the younger hypertensive group compared with the younger normotensive group, whereas this was not in the case among the elderly participants.

3.2. Impact of sleeping pill use on blood pressure in participants with no intake of antihypertensive medication

Table 3 presents characteristics and sleep measurements, including quantity and quality of nonuse low- and high-frequency-use groups, in participants with no intake of antihypertensive medication. Mean age was highest in the high-frequency-use group. The proportion of females was lowest in the nonuse group. BMI was lower in both the low- and high-frequency-use groups. Global PSQI score and each component of PSQI except C3 and C7 were significantly higher in both the low- and high-frequency-use groups than in the nonuse group. Figure 2 shows negative dose–response relationships between the frequency of sleeping pill use and SBP in each age-divided subgroup. SBP was highest in the nonuse group, whereas it was lowest in the high-frequency-use group in each age-divided subgroup. Similar relationships were found between the frequency of sleeping pill use and both DBP and PP. The age- and multivariable-adjusted OR for SBP, DBP, and PP are presented in Table 4. In participants with no intake of antihypertensive medication, the odds of high SBP (OR, 0.65; 95% CI, 0.52–0.82; $P<.0005$), high DBP (OR, 0.58; 95% CI, 0.38–0.85; $P<.005$), and high PP (OR, 0.77; 95% CI, 0.64–0.93; $P<.01$) were significantly lower in the high-frequency-use group compared with the nonuse group. The odds of high DBP (OR, 0.59; 95% CI, 0.34–0.96; $P<.05$) was significantly lower in the low-frequency-use group.

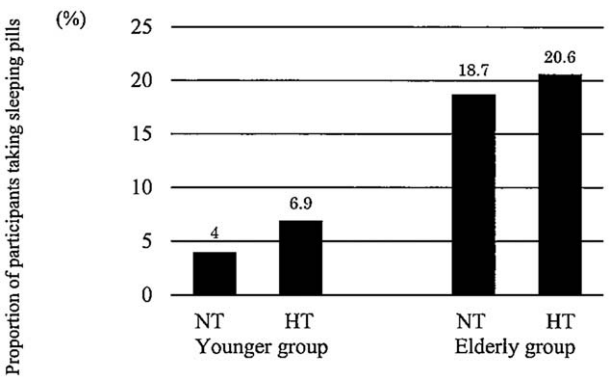

Figure 1. Proportion of sleeping pill use in normotensive and hypertensive participants. HT=hypertensive defined as intake of antihypertensive medication and/or systolic BP ≥140 mm Hg and/or diastolic BP ≥90 mm Hg; NT=normotensive; younger group was defined as age<65 years; elderly group was defined as age ≥65 years.

4. Discussion

In the present study, we investigated the association of hypertension with sleeping pill use and its effects on BP. First, the proportion of participants using sleeping pills was significantly higher in the hypertensive participants than in the normotensive ones. Second, among participants with no intake of antihypertensive medication, negative dose–response relationships between the frequency of sleeping pill use and SBP or DBP were found in each age-divided subgroup. Moreover, after suitable adjustment for confounding factors, the high-frequency sleeping pill use was significantly associated with lower odds of both high SBP and high DBP. Third, we found a lower rate of high PP in participants using sleeping pills.

In this study, we demonstrated that the proportion of participants using sleeping pills was significantly higher in the hypertensive participants than in the normotensive ones. Several studies have reported that sleep disorders and insomnia are associated with the development of hypertension.[1,2] Prejbisz et al[3] reported that a large proportion of patients with hypertension had insomnia. Because sleeping pills are commonly used for the treatment of sleep disorders and insomnia, our results are consistent with these previous observations. In addition, after adjusting for confounding variables, a significant difference was observed between hypertensive and normotensive participants in the younger group; however, no significant difference was found in the elderly group. Phillips et al[24] reported no significant association between insomnia and risk of hypertension in an elderly sample; whereas, they also reported that sleep complaints, including difficulty in falling asleep or waking up repeatedly during sleep, predicted an increased risk of hypertension in a middle-aged sample.[25] This suggested that the association between sleep disorders and hypertension differs with age, which, in this study, could have been mainly due to comorbidities, including diabetes and dyslipidemia, in particular; this is further supported by the fact that the statistically significant association between hypertension and sleeping pill use disappeared after including these comorbidities in the multivariate analysis.

In participants with no intake of antihypertensive medication, we observed negative dose–response relationships between the

Table 2

Univariate and multivariate OR for sleeping pill use (n = 11,225).

	Univariate			Multivariate model 1			Multivariate model 2		
	OR	95% CI	*P*	OR	95% CI	*P*	OR	95% CI	*P*
Hypertension									
All	1.68	(1.52–1.87)	<.0001	1.19	(1.06–1.33)	<.05	1.14	(1.02–1.28)	<.05
Age <65 y	1.80	(1.26–2.52)	<.005	1.59	(1.08–2.33)	<.05	1.49	(1.00–2.18)	<.05
Age ≥65 y	1.13	(1.01–1.26)	<.05	1.15	(1.02–1.30)	<.05	1.11	(0.98–1.25)	.09

Multivariate model 1 included age, gender, BMI, smoking, and drinking.
Multivariate model 2 included age, gender, BMI, smoking, drinking, diabetes, and dyslipidemia.
CI = confidence interval, OR = odds ratio.

frequency of sleeping pill use and SBP or DBP, leading to the following 2 hypotheses. First, sleeping pill use may improve sleep quality and elongate sleep duration, resulting in decreased BP, because both poor sleep quality and short sleep duration are involved in the activation of SNS and the elevation of BP[5–9]; however, in this study, the global PSQI score and each component of the PSQI, including C1, C2, C4, and C5 scores, were higher in both the low- and high-frequency-use groups than in the nonuse group, suggesting that participants using sleeping pills were suffering from poor sleep quality. Higher C2 (indicating long sleep latency) and C4 (indicating low sleep efficiency) scores could be associated with a percentage decrease of SWS and higher C5 score (indicating sleep disturbances) usually indicates an increase in sleep fragmentation. Percentage decrease of SWS increases the SNS activity and is involved in BP elevation.[26,27] Similarly, sleep fragmentation increases the SNS activity and adrenocortical activity.[28,29] Despite of these potential sleep quality-related BP elevation factors, SBP and DBP were lower in both the low- and high-frequency-use groups than in the nonuse group. Furthermore, we did not find a significant difference in C3 score (a marker of subjective sleep duration) between the nonuse group and low- or high-frequency-use groups, suggesting that sleep duration did not contribute to the effects of sleeping pills on BP. Effects of sleeping pills on sleep quality and sleep duration could not explain the low level of BP in participants taking sleeping pills in this study. Second, sleeping pill use may reduce BP, independent of their effects on sleep. Recent studies have reported that a new generation of sleeping pills, including melatonin receptor agonists and orexin receptor antagonists, may have beneficial effects on BP[12,13]; however, considering our study period, our results were based mainly on benzodiazepines or nonbenzodiazepines, including zolpidem, zopiclone, and eszopiclone. Experimental studies have shown that benzodiazepines can decrease BP and SNS activity not only by intravenous injection[14,15] but also by oral administration.[16] Zolpidem, a nonbenzodiazepine, can reduce SNS activity and nocturnal BP in nondipping hypertensive patients.[17] Therefore, the use of such sleeping pill may reduce BP, independent of its effects on sleep.

In addition, sleep disorders are often accompanied by anxiety,[30] and sleep disturbances may contribute to the development of anxiety and related disorders.[31] It has been reported that anxiety is involved in autonomic nervous dysfunction,[32] BP elevation, and the development of hypertension.[10,11] Antianxiety treatment using benzodiazepine effectively lowered BP in hypertensive patients.[33] Therefore, sleeping pill use may have beneficial effects on BP by reducing anxiety that frequently accompanies sleep disorders.

Interestingly, we found a negative dose–response relationship between the sleep pill use and PP. PP reflects stiffness of the aorta

Table 3

Characteristics of participants with no intake of antihypertensive medication.

	All	Nonuse	Low frequency	High frequency
n	7788	6869	344	575
Mean age, y	63.7 ± 16.0	62.5 ± 16.2	70.5 ± 12.0*	74.1 ± 10.2*
Female, n (%)	3655 (47)	3071 (45)	227 (66)	357 (62)
BMI, kg/m^2	22.5 ± 3.1	22.6 ± 3.1	22.1 ± 3.1†	21.9 ± 3.0*
Diabetes, n (%)	532 (7)	441 (6)	26 (8)	65 (11)
Dyslipidemia, n (%)	1065 (14)	844 (12)	83 (24)	138 (24)
Current smoker, n (%)	1070 (14)	1003 (15)	20 (6)	47 (8)
Habitual drinker, n (%)	2209 (28)	1985 (29)	84 (24)	140 (24)
Global PSQI score	4.9 ± 2.8	4.5 ± 2.4	7.6 ± 3.0*	8.8 ± 3.1*
C1 score	1.1 ± 0.6	1.0 ± 0.6	1.4 ± 0.6*	1.3 ± 0.7*
C2 score	0.8 ± 0.9	0.7 ± 0.8	1.4 ± 0.9*	1.3 ± 1.1*
C3 score	1.3 ± 0.9	1.3 ± 0.9	1.4 ± 0.9	1.2 ± 0.9
C4 score	0.4 ± 0.8	0.3 ± 0.7	0.7 ± 1.0*	0.7 ± 1.0*
C5 score	0.7 ± 0.5	0.7 ± 0.5	0.9 ± 0.5*	0.9 ± 0.6*
C6 score	0.3 ± 0.8	0.0 ± 0.0	1.4 ± 0.5*	3.0 ± 0.0*
C7 score	0.4 ± 0.6	0.4 ± 0.6	0.4 ± 0.7	0.4 ± 0.7

Nonuse, participants who did not use sleeping pills during the past month; low frequency, participants who took sleeping pills ≤2 days per week; data are expressed as mean ± standard deviation or number and percentage.
BMI = body mass index, PSQI = Pittsburgh Sleep Quality Index.
* $P < .0001$ versus the nonuse group.
† $P < .01$ versus the nonuse group.

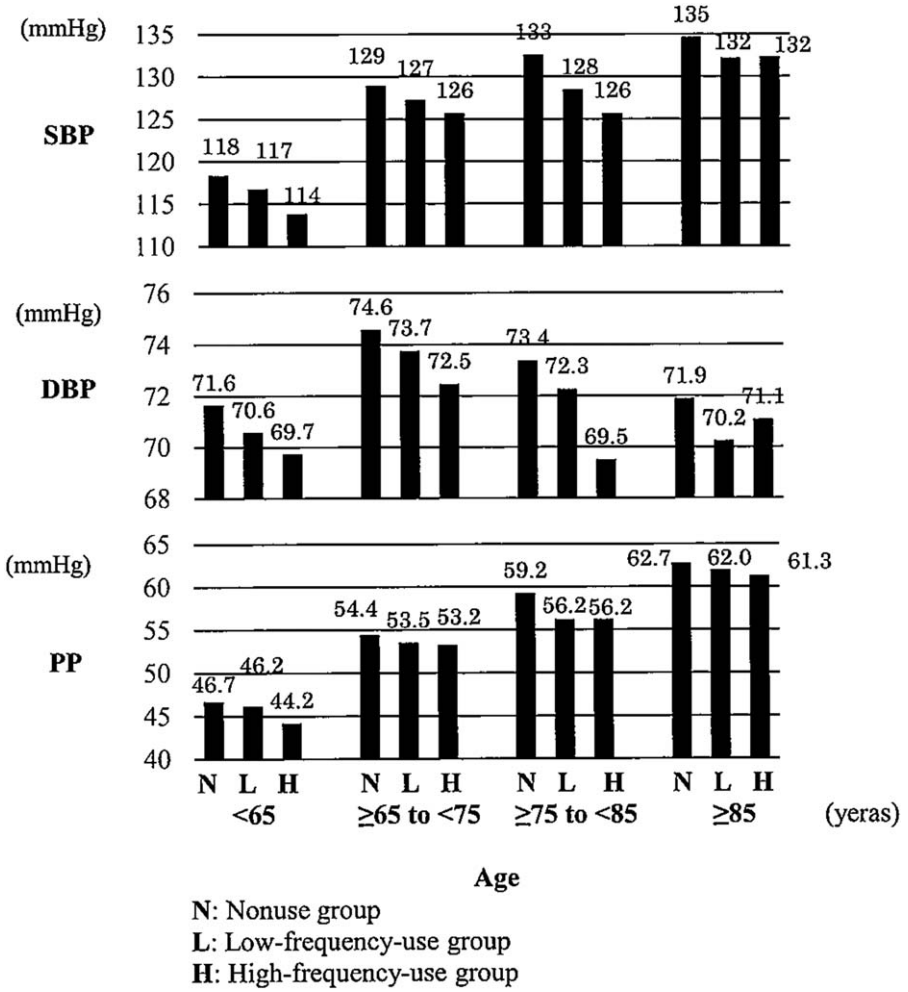

Figure 2. Association between the frequency of sleeping pill use and blood pressure and pulse pressure in each age group. SBP = systolic blood pressure, DBP = diastolic blood pressure, PP = pulse pressure; nonuse group was defined as participants who did not use sleeping pills during the past month; low-frequency-use group was defined as participants who took sleeping pills ≤2 days per week; high-frequency-use group was defined as participants who took sleeping pills ≥3 days per week.

Table 4

Age- and multivariable-adjusted OR for high BP and PP according to sleeping pill use.

	Age-adjusted			Multivariable-adjusted		
Sleeping pill use	**OR**	**95% CI**	***P***	**OR**	**95% CI**	***P***
High SBP						
Nonuse	1			1		
Low frequency	0.79	(0.59–1.03)	.08	0.88	(0.66–1.15)	.35
High frequency	0.60	(0.48–0.75)	<.0001	0.65	(0.52–0.82)	<.0005
High DBP						
Nonuse	1			1		
Low frequency	0.51	(0.30–0.83)	.005	0.59	(0.34–0.96)	.03
High frequency	0.51	(0.33–0.74)	<.0005	0.58	(0.38–0.85)	<.005
High PP						
Nonuse	1			1		
Low frequency	0.82	(0.65–1.03)	.09	0.88	(0.70–1.11)	.29
High frequency	0.74	(0.61–0.88)	<.005	0.77	(0.64–0.93)	<.01

Nonuse, participants who did not use sleeping pills during the past month; low frequency, participants who took sleeping pills ≤2 days per week; high frequency, participants who took sleeping pills ≥3 days per week.

Multivariable-adjusted model included age, gender, BMI, smoking, drinking, diabetes, and dyslipidemia.

BMI = body mass index, BP = blood pressure, CI = confidence interval, DBP = diastolic BP, OR = odds ratio, PP = pulse pressure, SBP = systolic BP.

and large arteries, which increase with arteriosclerotic change, and is closely related to hypertension.[34] Our findings could lead to the hypothesis that sleeping pill use may have beneficial effects on PP by decreasing BP; however, several previous studies have suggested that sleeping pill use may have adverse effects on health.[35] In particular, an increased relative risk for cardiovascular events was reported in participants receiving psychotropic drugs.[36] Our results seem inconsistent with these previous studies, because PP is an independent predictor of the risk of cardiovascular disease, especially coronary heart disease.[37] One possible explanation is that sleep disorders and insomnia may increase the risk for cardiovascular diseases,[38] which may have nullified the beneficial effects of sleeping pills in previous studies. Another explanation is that sleeping pill use could cause excessive fall in nocturnal BP, which may lead to reactive elevation of BP in the morning. The morning hypertension is one of the risk factors for cardiovascular disease.[39] Furthermore, sleeping pill use could cause residual daytime sleepiness, and impairment of psychomotor and cognitive functioning, rendering risks for accidents such as traffic accidents, falls and hip fractures.[40] Considering these aspects, it may not be advisable to use sleeping pills expecting the beneficial effects on BP and PP as presented in the present study.

This study had several limitations. First, due to its cross-sectional study design, a causal relationship between sleeping pill use and antihypertensive medication, BP, or PP cannot be predicted. Second, the types of antihypertensive agents used were not assessed in this study, although certain types of antihypertensive agents such as β-blockers may have adverse effects on sleep quality[41]; however, this did not have a considerable impact on our results. As first-line drugs for hypertension, calcium channel blockers, angiotensin II receptor antagonists, angiotensin-converting enzyme inhibitors, and diuretics are recommended according to the Japanese Society of Hypertension Guidelines for the Management of Hypertension 2014. β-Blockers are recommended only in patients with coronary heart disease, heart failure, or tachycardia.[20] Because this study excluded participants who underwent treatment for, or had a history of, cardiovascular disease, the rate of β-blocker use in the treatment for hypertension was much lower than that of other antihypertensive agents. Third, we did not assess the types of sleeping pills used in this study. Therefore, we could not provide the difference in impacts on BP between benzodiazepines and nonbenzodiazepines, although the different mechanisms to improve sleep may lead to different impacts on BP. Finally, we could not exclude the possibility that our study population included participants with obstructive sleep apnea (OSA), a risk factor for hypertension, because we did not assess OSA in this study; however, considering the low risk of OSA in nonobese individuals,[42] we believe that OSA had a limited influence on the association between sleeping pill use and BP in this study. Indeed, the proportion of obese individuals (defined as a BMI of $\geq 30\,kg/m^2$) in our sample was very small (2.6%).

In conclusion, our cross-sectional analysis demonstrated that sleeping pills were more frequently required in hypertensive participants than in their normotensive counterparts. Sleeping pill use may decrease BP and assist in the treatment of high BP in hypertensive patients with sleep disturbances. Assessments of sleep parameters, including sleep quality and quantity, could not explain these effects. Further studies are needed to clarify the underlying mechanisms between sleeping pills and BP or PP.

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