Insomnia and its association with hypertension in a community-based population in China: a cross-sectional study

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ABSTRACT

Objective To investigate the prevalence of hypertension and its association with insomnia in a community-based population in China.

Methods A cross-sectional study which recruited 10 054 participants aged ≥18 years was conducted in Beijing. The association between self-reported insomnia and hypertension was determined by multiple logistic regression models. Age, gender, education, obesity, body mass index, physical activity, current smoking, current drinking, work stress, diabetes and dyslipidaemia were adjusted for as confounders. Prevalence ratios (PRs) with corresponding 95% CIs were reported as effect measurements.

Results The number of subjects with no insomnia, occasional insomnia and frequent insomnia was 7632 (75.9%), 1545 (15.4%) and 877 (8.7%), respectively. The prevalence of hypertension in those with no insomnia, occasional insomnia and frequent insomnia was 37.3%, 43.0% and 48.0%. Compared with subjects with no insomnia, the multivariate adjusted PRs and 95% CIs for those with occasional insomnia and frequent insomnia were 1.01 (0.91 to 1.12) and 0.92 (0.83 to 1.03) for men and 1.08 (1.00 to 1.16) and 1.12 (1.02 to 1.22) for women.

Conclusions Self-reported insomnia is associated with a higher risk of hypertension in women.

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INTRODUCTION

Insomnia is a subjective feeling of having difficulty initiating or maintaining sleep, or having poor sleep quality.[1] It has been reported that the prevalence of insomnia is 9.2% in mainland China,[2] 39.2% in Hong Kong[3] and 25% in Taiwan.[4] In the UK the prevalence of insomnia symptoms has been reported as 35–38.6%.[5] In Canada, 40.2% of a study population presented at least one symptom of insomnia.[6] Several studies have found that insomnia is associated with substantial impairments of an individual's quality of life,[7] mental health[8] and accident occurrence.[9] Additionally, recent studies in industrialised countries have shown that insomnia is associated with myocardial infarction[10] and heart failure.[11] Previous studies also suggested that insomnia was more frequent in women than in men,[12] and gender discrepancy has been reported in the association between insomnia and heart failure.[11] It would therefore be reasonable to examine whether gender modifies the association between insomnia and hypertension and to stratify the analysis by gender.

Studies exploring the association between insomnia and hypertension have reported modest and inconsistent results. However, the association between self-reported insomnia and hypertension has hardly been examined in the Chinese population. In the present study we investigated the association between self-reported insomnia and hypertension in China, taking into account several established risk factors of hypertension based on a population-based survey.

MATERIALS AND METHODS

Study design and participants

This survey was a cross-sectional chronic diseases and risk factors study conducted in Beijing in 2007. Citizens or permanent residents (those who are registered Beijing citizens excluding those who lived outside Beijing for ≥6 months and non-registered Beijing citizens who have a temporary residence permit and have lived in Beijing for ≥6 months) who were aged ≥18 years were recruited using a multistage stratified random sampling design. We selected the first sampling unit according to the per capita gross domestic product of each district followed by the second sampling unit (towns and urban neighborhoods). Urban neighbourhood communities and villages were then selected as the final sampling unit and the final study population was recruited from the final sampling unit (communities). Two urban administrative districts, one urban–rural mixed district and one rural district were selected and 38 communities were then randomly sampled (see online supplementary file for flow chart). Mainland China has a unique residence registration system through which we were able to obtain the basic demographic information such as name, birth date and gender from the departments of local governments. Before the survey we informed local administrators of the aim and method of our study and, with their help, we were able to disseminate the study design via broadcasting and booklets. On the night before the survey the residents were told not to drink or eat from 20:00 to 08:00 the following day. The response rate was 83.5%.

Data collection

The health interview was performed by trained medical staff at community health centres using a well-established questionnaire to determine demographic and behavioural characteristics of the study population. Demographic information included birth date, gender and education and behavioural

information included current smoking status, current drinking status and physical activities. Education level was categorised as elementary school or lower (<7 years), middle or high school (7 – 12 years) and college or higher (>12 years).

Physical examination included anthropometric measurements, blood pressure, medical history and drug administration history. Height and weight were measured to the nearest 0.1 cm and 0.1 kg, respectively, with the subject standing barefoot in light clothes. Waist circumference was measured to the nearest 0.1 cm at the mid-point between the 12th rib and the right anterior superior iliac spine. Body mass index (BMI) was calculated as weight (kg) divided by the height squared (m^2). Blood pressure was measured using a standard mercury sphygmomanometer on the right arm in the sitting position after the participants had rested for 5 min. Phase 1 and phase 5 Korotkoff sound was used as systolic blood pressure (SBP) and diastolic blood pressure (DBP), respectively. Blood pressure was measured twice and the average results were used for data analysis. Medical history and drug administration history were obtained from medical records and confirmed by general practitioners. All the measurements were adopted by community licensed physicians.

Blood samples were collected from all the participants after overnight fasting. Biochemical measurements were conducted in the central laboratory of Peking University People's Hospital. Concentrations of fasting glucose, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG) and low-density lipoprotein cholesterol (LDL-C) were measured using an autoanalyser (Hitachi 717, Hitachi Instruments, Tokyo, Japan).

Diabetes mellitus was defined as fasting glucose ≥7.0 mmol/L or current medication for diabetes. Dyslipidaemia was defined as any of the following: TC >5.18 mmol/L, LDL-C >3.37 mmol/L, HDL-C <1.04 mmol/L or TG >1.70 mmol/L. Obesity was defined as waist circumstance >90 cm in men and >85 cm in women. A history of coronary heart disease or stroke, including acute myocardial infarction, angina pectoris and all other ischaemic heart disease, was obtained via medical records and reviewed by a cardiologist and a general practitioner.

Determination of self-reported insomnia and hypertension

Self-reported insomnia was collected via a question: "During the last month, have you had insomnia (eg, difficulty falling asleep or non-restorative sleep)?" with the following response options: no insomnia, occasional insomnia (1–2 times/week), frequent insomnia (≥3 times/week). Hypertension was defined as SBP ≥140 mm Hg, DBP ≥90 mm Hg or current medication for hypertension.

Statistical analysis

Continuous variables were presented as mean±SD and categorical variables were presented as frequencies and proportions. In the descriptive analysis we present the basic characteristics of study subjects and the prevalence of hypertension by insomnia. The Cochran–Armitage method was used to test for trend. In an exploratory analysis we examined the association between insomnia and hypertension using multiple logistic regression models in both men and women together. We also examined the interaction terms and found that there was an interaction effect between gender and self-reported insomnia for hypertension. The association in men and women separately was also examined. Three models were used for the analysis. The first model included only insomnia, the second model was adjusted for age (plus gender for both genders together) as confounders and the third model was adjusted for age, BMI, education, current smoking, current drinking, work stress, physical activity, obesity, diabetes and dyslipidaemia as confounders (plus gender for both genders together). We also examined the association between insomnia and previously diagnosed/undiagnosed hypertension and hypertension under poor/good control. Prevalence ratios (PRs) with 95% CIs were presented and $p<0.05$ was considered to be statistically significant. All of the statistical analyses were conducted using R 2.15.[13]

RESULTS

The number of subjects with no insomnia, occasional insomnia and frequent insomnia was 7632 (75.9%), 1545 (15.4%) and 877 (8.7%), respectively. Table 1 shows the basic characteristics of the participants by insomnia status. The mean±SD age was 51.8±13.4, 54.0±12.5 and 56.5±12.3 years for subjects with no insomnia, occasional insomnia and frequent insomnia, respectively. Of the 10 054 participants, 3935 (39.1%) had hypertension. As shown in table 2, the prevalence of hypertension for those with no insomnia, occasional insomnia and frequent insomnia was 40.5%, 40.0% and 41.4% in men and 35.3%, 44.0% and 50.2% in women. The Cochran–Armitage test for trend for the prevalence of hypertension among participants with different insomnia statuses was $p<0.0001$ for both genders. These trend tests were not consistent in men ($p=0.8840$) and women ($p<0.0001$).

Table 3 presents the PRs for hypertension in relation to insomnia. Model 1 included only insomnia status, model 2 was adjusted for age (plus gender when men and women were analysed together) and model 3 was further adjusted for several other confounders (education, BMI, physical activity, current smoking, current drinking, obesity, work stress, diabetes, and dyslipidemia). Compared with those without insomnia, the corresponding PRs and 95% CI for subjects with occasional insomnia and frequent insomnia were 1.01 (0.91 to 1.12) and 0.92 (0.83 to 1.03) for men and 1.08 (1.00 to 1.16) and 1.12 (1.02 to 1.22) for women after multivariable adjustments.

Table 4 shows the PRs for diagnosed hypertension for people with different insomnia status. The PRs for occasional insomnia and frequent insomnia were 1.04 (0.94 to 1.15) and 1.13 (1.00 to 1.29) for men and 1.03 (0.96 to 1.10) and 1.08 (1.02 to 1.14) for women after multivariable adjustment. The associations between insomnia and hypertension control are shown in table 5. The risk for poor hypertension control was 23% (PR 1.23, 95% CI 1.06 to 1.42) higher for men with frequent insomnia than for those with no insomnia. Likewise, women with frequent insomnia had a 18% (PR 1.18, 95% CI 1.05 to 1.32) higher risk of poor hypertension control compared with those with no insomnia.

DISCUSSION

Although many studies have focused on sleep disorders or hypertension, to our knowledge few trials have examined self-reported insomnia and hypertension in a single study and explored the association between them in the Chinese population.[14] In the present study we investigated the association of insomnia and hypertension in a community-based population and found that frequent insomnia in women was associated with a higher risk of hypertension than in those who reported no insomnia. This association was not statistically significant in men and was independent of age, education, BMI, physical activity, current smoking, current drinking, obesity, diabetes and dyslipidaemia.

Table 1 Basic characteristic of the study participants

	No insomnia (n=7632)	Occasional insomnia (n=1545)	Frequent insomnia (n=877)
Age (years)	51.8±13.4	54.0±12.5	56.5±12.3
BMI (kg/m^2)	25.4±3.9	25.3±4.0	25.0±3.8
Waist (cm)	87.0±10.9	86.3±10.5	86.3±10.6
SBP (mm Hg)	127.0±19.3	127.5±19.5	128.1±19.3
DBP (mm Hg)	80.2±10.5	79.5±10.4	79.8±10.6
Glucose (mmol/L)	5.08±1.67	5.16±1.79	5.18±1.69
TC (mmol/L)	4.82±0.96	4.91±1.00	4.99±1.03
TG (mmol/L)	1.53±1.48	1.51±1.39	1.49±1.14
LDL-C (mmol/L)	2.50±0.69	2.54±0.69	2.58±0.68
HDL-C (mmol/L)	1.32±0.34	1.34±0.30	1.35±0.32
Gender			
Men, n (%)	3065 (40.2)	400 (25.9)	222 (25.3)
Women, n (%)	4567 (59.8)	1145 (74.1)	655 (74.7)
Education			
0–6 years, n (%)	2061 (27.0)	510 (33.0)	353 (40.3)
7–12 years, n (%)	4698 (61.6)	881 (57.0)	445 (50.7)
>12 years, n (%)	873 (11.4)	445 (10.0)	79 (9.0)
Physical activity			
No, n (%)	3515 (46.1)	701 (45.4)	378 (43.1)
Yes, n (%)	4114 (53.9)	843 (54.6)	499 (56.9)
Current smoking			
No, n (%)	5015 (65.7)	1137 (73.6)	614 (70.0)
Yes, n (%)	2617 (34.3)	407 (26.4)	263 (30.0)
Current drinking			
No, n (%)	5855 (76.7)	1295 (83.8)	735 (83.8)
Yes, n (%)	1776 (23.3)	250 (16.2)	142 (16.2)
Work stress			
Low, n (%)	982 (12.9)	166 (10.8)	92 (10.5)
Intermediate, n (%)	4242 (55.6)	764 (49.5)	375 (42.9)
High, n (%)	2406 (31.5)	614 (39.8)	408 (46.6)
Obesity			
No, n (%)	4037 (52.9)	806 (52.2)	468 (53.4)
Yes, n (%)	3595 (47.1)	739 (47.8)	409 (46.6)
Diabetes			
No, n (%)	6796 (89.0)	1355 (87.7)	766 (87.3)
Yes, n (%)	836 (11.0)	190 (12.3)	111 (12.7)
Dyslipidaemia			
No, n (%)	3623 (47.5)	701 (45.4)	255 (40.5)
Yes, n (%)	4008 (52.5)	844 (54.6)	522 (59.5)
Stroke			
No, n (%)	7293 (95.6)	1476 (95.6)	803 (91.6)
Yes, n (%)	338 (4.4)	68 (4.4)	74 (8.6)
Coronary heart disease			
No, n (%)	7044 (92.3)	1354 (87.7)	708 (80.7)
Yes, n (%)	588 (7.7)	190 (12.3)	169 (19.3)

BMI, body mass index; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides.

Table 2 Prevalence of high SBP, high DBP and hypertension among subjects with different self-reported insomnia

	SBP ≥140 mm Hg	DBP ≥90 mm Hg	Hypertension
All			
No insomnia	1844 (24.2)	1616 (21.2)	2850 (37.3)
Occasional insomnia	389 (25.2)	315 (20.4)	664 (43.0)
Frequent insomnia	260 (29.6)	189 (21.6)	421 (48.0)
p Value*	0.0001	0.9185	<0.0001
Men			
No insomnia	797 (26.0)	811 (26.5)	1240 (40.5)
Occasional insomnia	90 (22.5)	103 (25.8)	160 (40.0)
Frequent insomnia	57 (25.7)	49 (22.1)	92 (41.4)
p Value*	0.4024	0.1811	0.8840
Women			
No insomnia	1047 (22.9)	805 (17.6)	1610 (35.3)
Occasional insomnia	299 (26.1)	212 (18.5)	504 (44.0)
Frequent insomnia	203 (31.0)	140 (21.4)	329 (50.2)
p Value*	<0.0001	0.0250	<0.0001

Values shown are n (%).
*Cochran–Armitage trend test.
DBP, diastolic blood pressure; SBP, systolic blood pressure.

The prevalence of self-reported frequent insomnia in our study was 8.7%, which was similar to that in another survey conducted in Beijing which reported a 9.2% prevalence of insomnia in the general population aged ≥18 years.[2] When occasional insomnia was included the prevalence was 24.1%, which was close to that in Taiwan[4] and lower than that in Hong Kong.[3] In Japan around 21.4% of adults had symptoms of insomnia,[15] which was comparable to the finding of 22.8% in South Korea.[16] Likewise, a study in Greece reported that 25.3% of subjects had insomnia[17] and, in Sweden, 32.1% of a study sample reported more than one symptom of insomnia.[18] Differences in sampling design methods, distribution of risk factors, socioeconomic status and daily lifestyle may partly explain these variations.

It has been pointed out that the prevalence of insomnia depends on how it is defined.[1] Currently, three main definitions of insomnia are widely used in the literature: the International Classification of Sleep Disorders (ICSD-2),[19] the Diagnostic and Statistics Manual (DSM IV-TR)[20] and the International Classification of Disease (ICD-10).[21] In our study, insomnia was measured using a single self-reported question rather than several more complicated questions pertaining to sleep duration, frequency or aetiology. Self-reported measures of health have been used in medical research for more than 20 years[22 23] and are widely used in medical research, especially in public health and epidemiology such as health behaviour and chronic disease surveillance.[24 25] It is also believed to be valid for measuring mental diseases such as insomnia, which is difficult to quantify.[26] Additionally, the single self-reported insomnia question used in our study reflected the understanding of insomnia by the study subjects and how they rated themselves rather than by three parties (including a physician or psychologist). It was therefore a useful subjective measure of insomnia.

Previous studies have suggested that women are more prone to insomnia than men,[12 27] and gender has also been found to play a role in cardiovascular disease risk and mortality.[28] A gender difference in the association between self-reported insomnia and hypertension might therefore be plausible. In our study the prevalence of self-reported insomnia was higher in women than in men (p<0.0001) but the prevalence of hypertension in women was not significantly different from that in men (p=0.1467). We therefore hypothesised that the association between insomnia and coronary heart disease might be modified by gender, and our findings did not contradict this hypothesis. The association between self-reported insomnia and hypertension was significantly different in men and women. PRs

Table 3 Association between self-reported insomnia and high SBP, high DBP and hypertension (PR (95% CI))

	Model 1	Model 2	Model 3
SBP ≥140 mm Hg			
All			
No insomnia	1	1	1
Occasional insomnia	1.04 (0.95 to 1.15)	0.98 (0.90 to 1.06)	0.99 (0.91 to 1.07)
Frequent insomnia	1.23 (1.10 to 1.37)	1.04 (0.94 to 1.15)	1.03 (0.94 to 1.14)
Men			
No insomnia	1	1	1
Occasional insomnia	0.87 (0.71 to 1.05)	0.88 (0.73 to 1.05)	0.92 (0.79 to 1.09)
Frequent insomnia	0.99 (0.78 to 1.24)	0.87 (0.70 to 1.09)	0.90 (0.73 to 1.11)
Women			
No insomnia	1	1	1
Occasional insomnia	1.14 (1.02 to 1.27)	1.02 (0.92 to 1.13)	1.02 (0.93 to 1.12)
Frequent insomnia	1.35 (1.19 to 1.53)	1.13 (1.02 to 1.26)	1.09 (1.00 to 1.20)
DBP ≥90 mm Hg			
All			
No insomnia	1	1	1
Occasional insomnia	0.96 (0.86 to 1.07)	1.01 (0.91 to 1.13)	1.01 (0.92 to 1.12)
Frequent insomnia	1.02 (0.89 to 1.16)	1.05 (0.92 to 1.21)	1.08 (0.95 to 1.23)
Men			
No insomnia	1	1	1
Occasional insomnia	0.97 (0.82 to 1.16)	0.97 (0.82 to 1.16)	1.00 (0.94 to 1.18)
Frequent insomnia	0.83 (0.65 to 1.08)	0.84 (0.65 to 1.08)	0.92 (0.72 to 1.17)
Women			
No insomnia	1	1	1
Occasional insomnia	1.05 (0.92 to 1.20)	1.03 (0.90 to 1.18)	1.00 (0.88 to 1.15)
Frequent insomnia	1.21 (1.03 to 1.42)	1.17 (0.99 to 1.37)	1.19 (1.02 to 1.39)
Hypertension			
All			
No insomnia	1	1	1
Occasional insomnia	1.15 (1.08 to 1.23)	1.06 (1.00 to 1.13)	1.05 (1.00 to 1.12)
Frequent insomnia	1.29 (1.19 to 1.39)	1.10 (1.02 to 1.18)	1.06 (1.01 to 1.13)
Men			
No insomnia	1	1	1
Occasional insomnia	0.99 (0.87 to 1.12)	1.00 (0.90 to 1.13)	1.01 (0.91 to 1.12)
Frequent insomnia	1.02 (0.87 to 1.20)	0.96 (0.82 to 1.11)	0.92 (0.83 to 1.03)
Women			
No insomnia	1	1	1
Occasional insomnia	1.24 (1.16 to 1.35)	1.09 (1.02 to 1.17)	1.08 (1.00 to 1.16)
Frequent insomnia	1.42 (1.31 to 1.55)	1.14 (1.07 to 1.21)	1.12 (1.02 to 1.22)

Model 1: insomnia.
Model 2: age, insomnia (plus gender when men and women analysed together).
Model 3: age, education, obesity, body mass index, physical activity, current smoking, current drinking, work stress, diabetes, dyslipidaemia, coronary heart disease, stroke, insomnia (plus gender when men and women analysed together).
DBP, diastolic blood pressure; PR, prevalence ratio; SBP, systolic blood pressure.

were higher in women than in men. Although the exact reason for the gender-specific differences in the association between insomnia and hypertension remains unclear, the results of recent publications are informative and persuasive. First, studies found that more women than men suffered from depression[29] and lacked the ability to use coping strategies,[30] both of which were related to insomnia.[31] Furthermore, sex hormones were reported to be of paramount importance in influencing gender differences in sleeping habits.[32] Additionally, an association between sleep deprivation and abnormal serum lipid levels was observed in women but not in men,[33] and dyslipidaemia was associated with a higher risk of hypertension in women.[34] Finally, a lower socioeconomic position for women might partly explain the discrepancy, but its contribution is limited.[35] In the present study, after adjusting for work stress, education and dyslipidaemia, the association between insomnia and hypertension was found only in women. This indicates that some other biological mechanisms account for the gender differences. This research question should be addressed extensively with more physiological markers in future large longitudinal epidemiological studies.

Several potential mechanisms have been proposed to explain the link between insomnia and hypertension. Insomnia is usually accompanied by short sleep duration. A recent meta-analysis reported that short sleep duration was associated with higher risks of hypertension.[36] Short sleep duration was also reported to increase BMI by reducing leptin and elevating ghrelin levels,[37 38] while higher weight and BMI increase the risk of hypertension over the life course.[39] In the present study the PRs of hypertension were still significant after adjusting for

Table 4 Association between self-reported insomnia and diagnosed hypertension (PR (95% CI))

Hypertension	Model 1	Model 2	Model 3
All			
No insomnia	1	1	1
Occasional insomnia	1.15 (1.09 to 1.22)	1.07 (1.01 to 1.13)	1.04 (1.00 to 1.11)
Frequent insomnia	1.16 (1.10 to 1.21)	1.11 (1.06 to 1.16)	1.08 (1.01 to 1.15)
Men			
No insomnia	1	1	1
Occasional insomnia	1.14 (1.02 to 1.26)	1.13 (1.02 to 1.26)	1.04 (0.94 to 1.15)
Frequent insomnia	1.30 (1.16 to 1.45)	1.24 (1.11 to 1.38)	1.13 (1.00 to 1.29)
Women			
No insomnia	1	1	1
Occasional insomnia	1.07 (1.00 to 1.14)	1.04 (0.98 to 1.11)	1.03 (0.96 to 1.10)
Frequent insomnia	1.12 (1.06 to 1.18)	1.10 (1.05 to 1.16)	1.08 (1.02 to 1.14)

Model 1: insomnia.
Model 2: age, insomnia (plus gender when men and women analysed together).
Model 3: age, education, obesity, body mass index, physical activity, current smoking, current drinking, work stress, diabetes, dyslipidaemia, coronary heart disease, stroke, insomnia (plus gender when men and women analysed together).
PR, prevalence ratio.

BMI and obesity. This indicates that the observed association is independent of BMI and obesity and has to be explained by other mechanisms. Although the pathophysiology of insomnia is not fully understood, it is generally regarded as a kind of hyperarousal disorder that is associated with increased activity of the hypothalamic-pituitary-adrenal-axis.[1] During this process, cortisol is excessively secreted and can be measured in both the plasma and urine.[40] Elevated cortisol is known to induce hypertension.[41 42] Finally, insomnia and hypertension may share some common risk factors such as smoking[43 44] and drinking alcohol.[3] However, after adjusting for these variables, the PRs only changed slightly. Since smoking and drinking were broadly measured in our study, residual confounding might explain the observed results. Thus, abnormalities of the neuroendocrine system and an unhealthy lifestyle may present a biologically plausible association between insomnia and hypertension. We also carried out an additional analysis to investigate the association between insomnia and previously diagnosed hypertension and poorly controlled hypertension and found that the associations between insomnia and hypertension seemed to be stronger for previously diagnosed hypertension and poorly controlled hypertension. These results suggest that people might be more prone to have insomnia if they know they have hypertension or poorly controlled hypertension. However, no longitudinal studies have been published regarding the incidence of insomnia after a diagnosis of hypertension.

Since the present data were collected using a cross-sectional design, we cannot make causal inferences as to whether insomnia precedes and causes hypertension. Furthermore, the potential non-response bias of the survey is of concern, as is the fact that more women than men participated in the survey. Women pay much more attention to their health, which could result in an overestimation of the prevalence of self-reported insomnia in the source population. Finally, insomnia was measured using a single question rather than the multiple questions used in other studies,[1 19–21] so we cannot evaluate the association between other insomnia symptoms and hypertension in the present study. The subjective measure of insomnia in the present study might be prone to misclassification of the exposure, which means the measurement of insomnia might include other aspects of mental disorders such as anxiety rather than insomnia per se. We acknowledge this limitation, although the same

Table 5 Association between self-reported insomnia and poor hypertension control (PR (95% CI))

Hypertension	Model 1	Model 2	Model 3
All			
No insomnia	1	1	1
Occasional insomnia	1.21 (1.06 to 1.37)	1.14 (1.00 to 1.30)	1.03 (0.96 to 1.10)
Frequent insomnia	1.27 (1.14 to 1.41)	1.21 (1.09 to 1.35)	1.08 (1.02 to 1.14)
Men			
No insomnia	1	1	1
Occasional insomnia	1.28 (1.02 to 1.60)	1.28 (1.03 to 1.60)	1.22 (0.97 to 1.53)
Frequent insomnia	1.46 (1.13 to 1.89)	1.43 (1.10 to 1.84)	1.23 (1.06 to 1.42)
Women			
No insomnia	1	1	1
Occasional insomnia	1.08 (0.93 to 1.25)	1.08 (0.93 to 1.25)	1.01 (0.87 to 1.17)
Frequent insomnia	1.19 (1.06 to 1.34)	1.19 (1.06 to 1.34)	1.18 (1.05 to 1.32)

Model 1: insomnia.
Model 2: age, insomnia (plus gender when men and women analysed together).
Model 3: age, education, obesity, body mass index, physical activity, current smoking, current drinking, work stress, diabetes, dyslipidaemia, coronary heart disease, stroke, insomnia (plus gender when men and women analysed together).
PR, prevalence ratio.

question about insomnia was addressed to all study participants so a non-differential misclassification would result in an underestimated effect size. The true estimate should be higher than reported.

CONCLUSIONS

Self-reported insomnia was associated with a higher risk of hypertension in this large Chinese sample. Future longitudinal studies should be conducted to further explore the causal relationship and to clarify the biological mechanisms between them.

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