Association between sleep quality and arterial blood pressure among Chinese nonagenarians/centenarians

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Summary

Background: There is association between sleep quality and arterial blood pressure, but it is still unclear if the association also exists in the very elderly. We examined the individual association between sleep quality and arterial blood pressure among the very elderly.

Material/Methods: The present study analyzed data from a survey that was conducted on all residents aged 90 years or older in a district with 2,311,709 inhabitants in 2005. Sleep quality was measured using The Pittsburgh Sleep Quality Index (PSQI).

Results: The subjects included in the statistical analysis were 216 men and 444 women. There were no significant differences in sleep quality scores, sleep latency, and sleep efficiency percentage and prevalence of poor sleep quality between subjects with and without hypertension. None of the differences in systolic blood pressure, diastolic blood pressure, and prevalence of hypertension, systolic hypertension and diastolic hypertension among subjects with well, fairly and poor sleep quality were significant. Multiple logistic regressions showed that unadjusted and adjusted Odds Ratio (ORs) of poor sleep quality for increased risk for hypertension were significant.

Conclusions: Among very elderly subjects, there was no association between sleep quality and arterial blood pressure.

key words: arterial blood pressure • sleep quality • nonagenarians/centenarians

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BACKGROUND

Many studies have established the association between sleep quality and arterial blood pressure. Subjects with chronic insomnia have a higher risk for hypertension [1–3] and U-shaped associations have also been demonstrated between sleep duration and hypertension [4]. 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 [5–8]. Given the well-established association of hypertension with significant medical morbidity (eg, hypertension, metabolic syndrome, osteoporosis), insomnia can induce hypertension through an activation of the hypothalamic-pituitary-adrenal (HPA) axis [9]. On the other hand, it has also been confirmed that subjects with hypertension are more likely to report emotional disorders such as anxiety, depression, and sleep disorders, which are usually viewed as one of their clinical features, indicating that hypertension is a risk factor for sleep disorders [10,11].

It is well known that both sleep quality and hypertension are strongly influenced by lifestyle [12]. Smoking and lack of exercise are linked with risk for hypertension or chronic insomnia [13,14]. It has been confirmed that there is a U-shaped association between alcohol consumption and hypertension or chronic insomnia [13,15]. Both sleep disorders and hypertension are associated with high mortality risk [4,16,19].

Insomnia is a risk factor for hypertension [1–3], and individuals with hypertension are more likely to have sleep disorders [10,11]. Some behaviors (eg, exercise, smoking, alcohol consumption) are risk factors for both sleep disorders and hypertension [13–15]; sleep disorders and hypertension are both risk factors for mortality in the elderly [4,16–19], and hypertension and sleep disorders are both related to age [20,21]. From all of these, we can conclude that in very elderly subjects (aged 90 years or more), there is a close association between sleep quality and arterial blood pressure, which may be different from that in the general population of older adults (aged 60 years or more). However, to our knowledge, no population-based study has evaluated the association between sleep quality and arterial blood pressure in the very elderly. In this study, we examined the association between sleep quality and arterial blood pressure in the very elderly subjects using data from a sample of Chinese nonagenarians and centenarians.

MATERIAL AND METHODS

Materials

The methods were reported previously [21]. In brief, on the basis of the Dujiangyan (located in Sichuan province, southwest China) 2005 census, a cross-sectional study for age-related diseases was conducted in 870 very elderly subjects (≥90 years), which was a part of the Project of Longevity and Aging in Dujiangyan (PLAD). The PLAD aimed to investigate the relationship between environments, lifestyle, genetics, longevity and age-related diseases. Volunteers were examined by trained professional physicians according to basic health criteria and the results were entered into standard forms, especially the questionnaire on lifestyles (including The Pittsburgh Sleep Quality Index (PSQI)). In this analysis, subjects were excluded who were using medicine for treating sleep problems of hypertension, were bedridden, had cancer, a history or clinical evidence of stroke, secondary hypertension, or were receiving anti-hypertensive treatment, had terminal stage of physical disease such as respiratory system disease, cardiovascular disease, kidney disease, dementia or cognitive decline. Overall, 21 men and 26 women were not eligible for the study because they had already died or moved away from the area. Of the 262 men and 561 women who were interviewed, 46 men and 117 women were excluded for the reasons above. The study population ultimately consisted of 660 very elderly subjects. Informed consents were obtained from all participants (as well as their legal proxies). The Research Ethics Committee of the Sichuan University approved the study. To assure reliability of this information, during the course of interviewing, at least one family member, who usually lived with the participant, took part in the interviewing and checked the completed questionnaire for accuracy.

Data collection and measurements

Measurement of blood pressure

The methods were reported previously [21]. Sitting or recumbent position, right arm blood pressure (BP) was measured twice to the nearest 2 mmHg using a standard mercury sphygmomanometer (phases I and V of Korotkoff) by trained nurses or physicians. The mean value of 2 measurements was used to calculate systolic BP (SBP) and diastolic BP (DBP), and the SBP and DBP were calculated as the mean of right and left arm values in exceptional subjects [22]. The mean of 2 readings was used for classification of BP according to Joint National Committee (JNC)VII criteria into normal (SBP ≤120 mmHg and DBP ≤80 mmHg), pre-hypertension (SBP 120–139 mmHg and/or DBP 80–89 mmHg), stage 1 hypertension (SBP 140–159 mmHg and/or DBP 90–99 mmHg), stage 2 hypertension (SBP ≥160 mmHg and/or DBP ≥100 mmHg), systolic hypertension was defined as a SBP >140 mmHg, the isolated systolic hypertension (ISH) was defined as a SBP >140 mmHg and a DBP <90 mmHg, diastolic hypertension was defined as a DBP >90 mmHg, and the isolated diastolic hypertension (IDH) was defined as a SBP <140 mmHg and a DBP >90 mmHg. Hypertension was defined as a SBP >140 mmHg and/or a DBP >90 mmHg and/or receiving anti-hypertensive treatment [22]. Subjects with confirmed hypertension and no identified cause of secondary hypertension were diagnosed with essential hypertension.

Measurement of sleep quality

The Pittsburgh Sleep Quality Index (PSQI) was used for the subjective assessment of sleep quality [23]. The PSQI is a questionnaire consisting of 19 items which are coded on a 4-point scale (0–3) to obtain 7 subcategories: sleep duration, sleep disturbances, sleep latency, daytime dysfunction, sleep efficiency, sleep satisfaction, and medication use. The sum of all subscores represents the total sleep quality score, ranging between 0–21, with higher scores representing lower sleep quality. The individuals were categorized as follows: good sleep quality (scores between 0 and 5); fairly good sleep quality (scores between 6 and 10); and poor sleep quality (scores between 11
and 21). Respondents are asked to rate their sleep reflecting on the past month. Psychometric properties have demonstrated good reliability (internal consistency: 0.89; test retest reliability: 0.85) and good construct validity for the English language version [24]. The PSQI is a widely used tool in research studies and clinical trials, and has been translated into several languages including Chinese, Spanish, German, and Hebrew, with comparable reliability and validity values. Internal consistency in the present study was α=0.69. When excluding the medication use subscale due to a low rate of medication users in this sample, internal consistency increased to α=0.76. Outcome measures included the total PSQI score as well as self-report of sleep latency (SL) (question 2: “How long (in minutes) has it usually taken you to fall asleep each night?”), and sleep efficiency percentage (SE), computed as the ratio between the hours of actual sleep (question 4: “How many hours of actual sleep did you get at night?”) and total time in bed (hours computed based on reported bedtime in question 1: “What time have you usually gone to bed at night?” and reported wake-time in question 3: “What time have you usually gotten up in the morning?”), multiplied by 100 [24,25].

Assessment of covariates

The baseline examination included information on age (years), sex (Male/Female), smoking habits (yes or no), alcohol consumption (yes or no), tea consumption (yes or no), exercise (yes or no), serum lipid/lipoprotein levels (including serum triglyceride (TG), total cholesterol (TC), high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol), fasting blood glucose (FBG), serum uric acid (SUA), and body mass index (BMI) [21,22]. The BMI was calculated as body weight in kilograms divided by height in meters squared. Serum lipid/lipoprotein levels, FBG and SUA were determined by standard laboratory techniques (performed by a technician in the biochemistry laboratory of Sichuan University). The other covariates were collected by using a general questionnaire.

Behaviors of smoking, alcohol consumption, tea consumption, and exercise, which included former and current status, were collected by using a general questionnaire. In the questionnaire, every item had 2 options (yes or no). We defined subjects with such behaviors as doing it almost everyday for more than 2 years as of a year before, were classified as those with these habits previously, otherwise as without. Alcohol consumption included spirits, liqueurs, wine, sherry, martini, beer, lager, hard cider, stout, and so on. Tea consumption included all types of tea.

Statistical analysis

All of the statistical analyses for this study were performed with the SPSS for Windows software package, version 11.5 (SPSS Inc, Chicago, Illinois, USA). Baseline characteristics were compared between subjects with and without hyper-tension, between subjects with and without systolic hypertension, between subjects with and without diastolic hypertension using Pearson chi-square or Fisher’s exact test (where an expected cell count was ≤5) for categorical variables and unpaired Student’s T test for continuous variables. Baseline characteristics were also compared among different sleep quality groups using one-way for analysis of variance (ANOVA) for continuous variables and Pearson chi-square or Fisher’s exact test (where an expected cell count was ≤5) for categorical variables. Multiple logistic regression was used to estimate the odds ratio (OR) and 95% confidence interval (CI) of sleep quality as a function of increased risk for hypertension, systolic hypertension or diastolic hypertension. The following items were adjusted: age, sex, serum Lipid/lipoprotein, body mass index, blood glucose level, smoking, alcohol consumption, tea consumption, and exercise. A P value <0.05 was considered to be statistically significant, and all of the P values were 2-sided.

RESULTS

Baseline characteristics, arterial blood pressure and sleep quality

Among the 660 participants, mean age was 93.52 years, 69 were centenarians and 444 were women. Ninety percent of participants lived in the countryside. The mean SBP and DBP in the population were 139.71 (s.d.: 23.31) mmHg and 72.59 (s.d.: 12.06) mmHg. There were 150 (22.7%) subjects with normal arterial blood pressure, 133 (20.1%) with pre-hypertension, 216 (32.7%) with stage 1 hypertension, 161 (24.5%) with stage 2 hypertension, 364 (55.2%) with systolic hypertension, and 86 (13.0%) with diastolic hypertension (Tables 1 and 2).

There were 385 (58.4%), 128 (19.4%) and 147 (22.2%) with good, fairly good, and poor sleep quality respectively. The subjects with good sleep quality were younger in age (93.32±3.52, 93.95±3.39, 93.63±3.14, among subjects with good, fairly good, poor sleep quality respectively, P=0.016). The mean sleep quality score was 6.8±2.15, the mean SL was 45.89±16.72 min, and the mean SE was 76.53±8.78% (Tables 1 and 2).

No association between arterial blood pressure and quality of sleep

Among the different sleep quality groups, none of the differences in levels of SBP, DBP, the prevalence of hypertension, systolic hypertension and diastolic hypertension was significant (SBP: 140.21±23.20, 138.35±22.24, 140.37±22.78, P=0.703; DBP: 72.77±12.30, 72.50±10.92, 72.23±12.65, P=0.889; hypertension: 57.66%, 56.25%, 57.14%, P=0.847; systolic hypertension: 55.32%, 55.47%, 54.42%, P=0.836; diastolic hypertension: 13.50%, 8.59%, 15.64%, P=0.334 among subjects with good, fairly good, and poor quality sleep, respectively) (Table 1).

Between subjects with and without hypertension, none of the differences in sleep quality scores, sleep latency, sleep efficiency percentage and prevalence of poor sleep quality was significant (sleep quality scores: 6.67±2.59 vs. 6.07±2.66, P=0.139; sleep latency: 44.10±13.17 vs. 47.25±18.11, P=0.235; sleep...
Table 1. Baseline characteristics according to Sleep quality (n=660).

| Characteristics | All (n=660) | Well sleep quality (n=385) | Fairly sleep quality (n=128) | Poor sleep quality (n=147) |
|----------------|------------|---------------------------|-----------------------------|--------------------------|
|                |            | χ² or t                   | P value                     | χ² or t                   | P value                     |
| Age (years)    | 93.52±3.37 | 93.32±3.32                | 93.95±3.59                  | 93.63±3.14                | 1.882                      | 0.016*                      |
| Gender         |            |                           |                             |                          |                            |                             |
| Male/Female    | 216/444    | 138/247                   | 36/92                       | 42/105                   | 3.964                      | 0.138                       |
| Hypertension   |            |                           |                             |                          |                            |                             |
| Yes/No         | 378/282    | 222/163                   | 72/56                       | 84/63                    | 0.809                      | 0.847                       |
| DBP(mmHg)      | 72.59±12.06| 72.77±12.30               | 72.50±10.92                 | 72.23±12.65              | 0.106                      | 0.889                       |
| DH (Yes/No)    | 86/574     | 52/333                    | 11/17                       | 23/124                   | 1.326                      | 0.334                       |
| SBP(mmHg)      | 139.71±23.31| 140.21±23.20            | 138.35±22.24                | 140.37±22.78             | 0.353                      | 0.703                       |
| SH (Yes/No)    | 364/296    | 213/172                   | 71/57                       | 80/67                    | 0.858                      | 0.836                       |
| TG (mmol/L)    | 1.22±0.64  | 1.23±0.70                 | 1.18±0.51                   | 1.25±0.58                | 0.405                      | 0.667                       |
| TC (mmol/L)    | 4.15±0.85  | 4.05±0.84                 | 4.09±0.86                   | 3.97±0.88                | 0.831                      | 0.436                       |
| HDL (mmol/L)   | 1.58±0.59  | 1.56±0.39                 | 1.57±0.35                   | 1.63±1.02                | 0.222                      |                             |
| LDL (mmol/L)   | 2.28±0.97  | 2.23±0.59                 | 2.30±0.64                   | 2.40±0.97                | 1.658                      | 0.191                       |
| FBG(mmol/L)    | 4.46±1.45  | 4.44±1.32                 | 4.40±1.48                   | 4.57±1.68                | 0.536                      | 0.585                       |
| SUA (µmol/L)   | 318.72±87.01| 318.34±84.83          | 316.75±83.66                | 321.45±93.10             | 4.993                      | 0.007**                     |
| BMI            | 19.05±3.64 | 19.02±3.25                | 18.91±3.85                  | 19.20±3.85               | 0.219                      | 0.803                       |

Baseline characteristics were compared between different arterial blood pressure groups. * P<0.05 ** P<0.01 vs. Non-cognitive impairment. Using χ² or Fisher’s exact test (where *** an expected cell count was <5) for categorical variables and unpaired Student’s t test for continuous variables.

In the testing, a P value <0.05 was considered to be statistically significant. Poor sleep quality; SL – sleep latency; SE – sleep efficiency percentage; SUA – Serum uric acid; BMI – body mass index; FBG – fasting blood glucose; HDL – high density lipoprotein; LDL – low density lipoprotein; TC – Total cholesterol; TG – Triglyceride; SH – systolic hypertension; DH – diastolic hypertension.

efficiency percentage: 79.17±12.64 vs. 74.56±14.36, P=0.473; prevalence of poor sleep quality: 22.34% vs. 22.22%, P=0.778 in subjects without and with hypertension, respectively). Between subjects with and without systolic hypertension, none of the differences in sleep quality scores, sleep latency, sleep efficiency percentage and prevalence of poor sleep quality was significant (sleep quality scores: 6.71±2.71 vs. 6.95±2.48, P=0.172; sleep latency: 44.35±13.42 vs. 47.14±17.99, P=0.347; sleep efficiency percentage: 79.21±11.91 vs. 74.35±13.44, P=0.469; prevalence of poor sleep quality: 22.64% vs. 21.98%, P=0.813 in subjects without and with systolic hypertension, respectively). Between subjects with and without diastolic hypertension, none of the differences in sleep quality scores, sleep latency, sleep efficiency percentage and prevalence of poor sleep quality was significant (sleep quality scores: 6.77±3.01 vs. 7.31±2.67, P=0.212; sleep latency: 45.99±13.57 vs. 45.22±11.65, P=0.881; sleep efficiency percentage: 76.34±2.25 vs. 77.79±2.81, P=0.786; prevalence of poor sleep quality: 26.74% vs. 21.60%, P=0.207 in subjects without and with diastolic hypertension, respectively).

Neither the unadjusted nor the adjusted multiple logistic regression showed that good quality sleep had a function of decreasing the risk for hypertension (unadjusted odds ratio (OR): 0.963, 95% confidence interval (CI): 0.654–1.417; adjusted OR: 0.981, 95% CI: 0.736–1.328), systolic hypertension (unadjusted OR: 0.968, 95% CI: 0.674–1.497; adjusted OR: 0.976, 95% CI: 0.615–1.541) or diastolic hypertension (unadjusted OR: 0.821, 95% CI: 0.543–1.205; adjusted OR: 0.904, 95% CI: 0.649–1.398) (Table 3). All of these showed that among very elderly subjects there was no association between sleep quality and arterial blood pressure.

**DISCUSSION**

This study evaluated the association between sleep quality and arterial blood pressure in very elderly subjects. In the present study, sleep quality included quality classification and scores, sleep latency and sleep efficiency. Arterial blood pressure included systolic blood pressure, diastolic blood pressure, hypertension, systolic hypertension and diastolic hypertension. In the cross-sectional observations, there was no association between sleep quality and arterial blood pressure.

Several studies have shown that there was a strongly significant association of sleep quality with arterial blood pressure,
Arterial blood pressure

Normal
SBP
DBP

Systolic hypertension

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study, participants were all aged 90–108 years. Our finding
in a population aged less than 60 years it was significant,
quality and arterial blood pressure changed with age; that
should conclude here that the association between sleep
previous studies and our finding in the present study, we
provided the evidence from very elderly subjects and ex

Baseline characteristics were compared between different arterial blood pressure groups. * P<0.05 ** P<0.01 vs. Normotension. Using χ² or Fisher’s exact test (where *** an expected cell count was <5) for categorical variables and unpaired Student’s t test for continuous variables. In the testing, a P value <0.05 was considered to be statistically significant. SUA – Serum uric acid; BMI – body mass index; FBG – fasting blood glucose; HDL – high density lipoprotein; LDL – low density lipoprotein; TC – Total cholesterol; TG – Triglyceride; SBP – systolic blood pressure; DBP – Diastolic blood pressure.

Table 3. Association between sleep and odds of hypertension.

| Characteristics       | Hypertension Unadjusted (95% CI) | Hypertension Adjusted (95% CI) | Systolic hypertension Unadjusted (95% CI) | Systolic hypertension Adjusted (95% CI) | Diastolic hypertension Unadjusted (95% CI) | Diastolic hypertension Adjusted (95% CI) |
|-----------------------|----------------------------------|--------------------------------|------------------------------------------|------------------------------------------|------------------------------------------|------------------------------------------|
| Sleep quality         |                                  |                                |                                          |                                          |                                          |                                          |
| Well                  | 0.963 (0.654–1.417)              | 0.963 (0.597–1.481)            | 0.968 (0.674–1.497)                     | 0.976 (0.615–1.541)                     | 0.917 (0.589–1.324)                     | 0.901 (0.571–1.248)                     |
| Fairly                | 1.008 (0.623–1.632)              | 0.806 (0.457–1.422)            | 1.013 (0.644–1.658)                     | 1.006 (0.636–1.358)                     | 0.867 (0.545–1.382)                     | 0.896 (0.512–1.541)                     |
| Poor                  | 1.000 (reference)                | 1.000 (reference)              | 1.000 (reference)                       | 1.000 (reference)                       | 1.000 (reference)                       | 1.000 (reference)                       |

OR – odds ratio. Unadjusted: Wald Chi-square test with df=1 was used; Adjusted multiple logistic regression was used to adjust for covariates, adjustment made with the followings: age, blood pressure, body mass index, blood glucose level, smoking habit, alcohol consumption, tea consumption, exercise.

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but that in a population aged more than 60 years (including general elderly aged 60 years or older and very elderly aged 90 years or older) it was non-significant.

Progressive arterial stiffness, as a result of structural and functional changes within the vessel wall, is a feature of aging and may precede the onset of hypertension [26–31]. Isolated systolic hypertension, defined as raised SBP but normal DBP, is the predominant form of hypertension in elderly people (265 years); systolic-diastolic hypertension is not as common in this population, as DBP stabilizes or even declines with advancing age [26,32–34]. In our study, in very elderly Chinese, there were high prevalences of hypertension (57.2%), systolic hypertension (55.2%), and isolated systolic hypertension (44.2%), and low prevalences of diastolic hypertension (13.0%), systolic-diastolic hypertension (11.0%) and isolated diastolic hypertension (2.0%). The prevalence of hypertension, systolic hypertension and isolated systolic hypertension in the present study was higher than that in the elderly aged 60 years or older from the previous reports, but the prevalence of diastolic hypertension, systolic-diastolic hypertension and isolated diastolic hypertension were lower than that in the elderly aged 60 years or older from the previous reports. This was consistent with previous findings that with advancing age, progressive arterial stiffness results in SBP increase and DBP decline. These indicate that systolic and diastolic blood pressure has different clinical significance in the elderly, especially in very elderly subjects. Considering this, we further independently assessed the association of systolic and diastolic blood pressure with sleep quality when the data were analyzed. All the results showed that there was no association between sleep quality and arterial blood pressure in the elderly subjects; this strongly confirmed the non-significant association between sleep quality and arterial blood pressure in very elderly subjects.

Among children, adolescents, and general adults, the association of sleep quality with hypertension could be explained by the following. Habitually sleep problems could lead to the development and maintenance of hypertension through elevated sympathetic nervous system activity, waking physical and psychosocial stressors, increased salt retention and changed exposure to raised 24-hour blood pressure and heart rate. Extended exposure to these forces could lead to the entrainment of the cardiovascular system to operate at elevated pressure equilibrium through structural adaptations such as arterial and left ventricle hypertrophic remodeling [30]. Habitual sleep problems could also contribute to hypertension by disrupting circadian rhythmicity and autonomic balance. Dramatic alterations in these parameters in modern industrialized society are theorized to cause the environment sensed by the brain to become metabolically flattened and arrhythmic, disturbing the circadian rhythmicity of blood pressure in susceptible individuals [35]. Hypertension is characterized by a disturbance in the circadian rhythmicity of many physiological variables, such as a shifting of the daily blood pressure profile to higher values, an increased prevalence of the non-dipping pattern, increased blood pressure variability, and disturbances in the diurnal rhythm of cardiac output [36]. Hypertensive subjects have been shown to have reductions of >50% in the 3 main neuronal populations of the SCN in comparisons between normal subjects and hypertensive subjects who died of myocardial infarction or brain hemorrhage [37].

There was the difference between the elderly and younger adults, children or adolescents, and there was no association of sleep with hypertension in the elderly. The mechanism of the difference is unclear and should be further explored, and might be related with the following. Firstly, there was a high mortality rate among the elderly, specifically among the every elderly, and both sleep problem and hypertension were related with high mortality rate. The high mortality in the elderly more likely removed those both with sleep problem and hypertension, and left the one or the other, and removed the association between sleep problem and hypertension. Secondly, degradation of arterial wall structure with age resulted in elevated arterial blood pressure, a high risk for hypertension, and insensitivity for sympathetic hormone. In the present study, there was a high prevalence of hypertension (57.19%) in the sample, and provide an evidence for a high prevalence of hypertension in the elderly [26,31–34]. Activation of the sympathetic nervous system is an important mechanism that links sleep disorders with combined systolic and diastolic hypertension, which is common in middle-aged hypertensive patients. However, no mechanistic link has been identified between sleep disorders and isolated systolic hypertension that results from age-dependent loss of arterial compliance and accounts for nearly 60% of hypertension in elderly populations [30–35]. Thirdly, advanced age is associated with changes in sleep architecture, with increased difficulties in sleep initiation and maintenance. Elderly subjects, who are often retired, also have more opportunities to take naps during the day [36–38].

Our study had some limitations that deserve mention. First, 870 subjects aged 90 years or older volunteered for the PLAD Study, and among these 870 volunteers only 660 were included in the statistical analysis. There might be selection biases. Because subjects with cancer, a history or clinical evidence of stroke, terminal stage of physical disease such as of respiratory system disease, cardiovascular disease, and kidney disease, usually have symptoms which could lead to insomnia and being bedridden. It is difficult to measure sleep quality of bedridden individuals. However, as our sample was from the community, not from hospital or nursing home, the information from them (excluded from the sample) might not be practice the practical implications of the present study. Second, because of the cross-sectional nature of this study, the subjects might change their diets and the conditions related to elevated hypertension and habits of sleep. However, the lifestyles, habits of sleep and food habits of the nonagenarians/centenarians were relatively stable and similar. Third, since this was a part of the PLAD, there might be a survival bias. However, this is inherent in any study of individuals of this age group. Finally, we did not adjust for other potential confounding factors, such as socio-economic status and family history of sleep disorders.

Conclusions

To our knowledge, this is the first study to demonstrate the association between sleep quality and arterial blood pressure in long-lived subjects, but the association was non-significant. This is different from that in children, adolescents,
and general adult (60 years or younger), and is similar with that in the general elderly (60 years or more). In very elderly Chinese, there was high prevalence of hypertension, especially systolic hypertension and isolated systolic hypertension.

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Conflicts of interest
None.

REFERENCES:

1. Stranges S, Dorn JM, Cappuccio FP et al: A population-based study of reduced sleep duration and hypertension: the strongest association may be in premenopausal women. J Hypertens, 2010; 28(5): 896–902
2. Phillips B, Mannino DM: Do insomnia complaints cause hypertension or cardiovascular disease? J Clin Sleep Med, 2007; 3: 489–94
3. Suka M, Yoshida K, Saginmori H: Persistent insomnia is a predictor of hypertension in Japanese male workers. J Occup Health, 2003; 45: 344–50
4. Gottlieb DJ, Redline S, Nieto FJ et al: Association of usual sleep duration with hypertension: the Sleep Heart Health Study. Sleep, 2006; 29: 1099–14
5. Vgontzas AN, Tsigos C, Bixler EO et al: Chronic insomnia and activity of the stress system: a preliminary study. J Psychosom Res, 1998; 45: 21–31
6. Vgontzas AN, Bixler EO, Lim HM et al: Chronic insomnia is associated with nocturnal activation of the hypothalamic-pituitary-adrenal axis: clinical implications. J Clin Endocrinol Metab, 2001; 86: 3787–94
7. Rodenburg A, Cohrs S, Jordan W et al: The sleep-improving effects of doxepin are paralleled by a normalized plasma cortisol secretion in primary insomnia. Psychopharmacology, 2003; 170: 423–28
8. Verkerreiser M, Van Dongen HP, Kerkhof GA: Physiologic indexes in chronic insomnia during a constant routine: evidence for general hyperarousal? Sleep, 2005; 28: 1588–96
9. Chronos GP: The role of stress and the hypothalamic-pituitary-adrenal axis in the pathogenesis of the metabolic syndrome: neuroendocrine and target tissue-related causes. Int J Obes, 2000; 24: 550–55
10. Gangwisch JE, Heymsfield SB, Boden-Albala B et al: Short Sleep Duration As a Risk Factor for Hypertension. Hypertension, 2006; 47: 833–39
11. Lopez-Garcia E, Faubel R, Guallar-Castillon P et al: Self-reported sleep duration and hypertension in older Spanish adults. J Am Geriatr Soc, 2009; 57(4): 663–68
12. Barbara P, Petra Bůžková, Paul E: Insomnia Did Not Predict Incident Hypertension in Older Adults in the Cardiovascular Health Study. Sleep, 2009; 32(1): 65–72
13. Julia FB, Tilen J, Arie KN et al: Sleep Duration and Hypertension Are Not Associated in the Elderly. Hypertension, 2007; 50: 385–89
14. Basle J: Hypertension in the elderly: a review of the importance of systolic blood pressure elevation. J Clin Hypertens (Greenwich), 2002; 4(2): 108–12
15. Greenwald SE: Ageing of the conduit arteries. J Pathol, 2007; 211(2): 157–72
16. Izzo JJ, Jr, Mitchell GF: Aging and arterial structure-function relations. Adv Cardiol, 2007; 44: 19–34
17. Tu LL, Bevers DG, Lip GY: Systolic vs diastolic blood pressure and the burden of hypertension.J Hum Hypertens, 2002; 16(5): 147–50
18. Kreier F, Vilmaz A, Kalbcek A et al: Hypothesis: shifting the equilibrium from activity to food leads to autonomic unbalance and the metabolic syndrome. Diabetes, 2003; 52: 2652–56
19. Coca A: Circadian rhythm and blood pressure control: physiological and pathophysiologic factors. J Hypertens, 1994; 12: 513–21
20. Goncharuk VD, Van Heerikhuize J, Duijnp et al: Neuropeptide changes in the suprachiasmatic nucleus in primary hypertension indicate functional impairment of the biological clock. J Comp Neurol, 2001; 431: 320–30
21. Haas DC, Foster GL, Nieto J et al: Age-dependent associations between sleep-disordered breathing and hypertension. Circulation, 2005; 111: 614–21