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ORIGINAL ARTICLE

Association between passive smoking and hypertension in Chinese non-smoking elderly women

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Although active smoking is linked with hypertension, the effect of environmental tobacco smoke exposure on hypertension has rarely been studied, and the scant epidemiological data available have yielded uncertain findings. Therefore, we conducted a cross-sectional study in a representative urban area of Beijing, China. A two-stage stratified clustering sampling method was performed, and 1078 female participants aged ⩾60 years were included in the analysis. Among the participants without antihypertensive treatment, >2 hours of daily passive cigarette smoking was significantly associated with higher systolic (by 4.24) and diastolic blood pressure (by 2.09) after multiple adjustments. The prevalence of hypertension was significantly higher in passive smokers (71.9%) than in non-passive smokers (66.1%). After adjusting for potential confounders, a positive association was observed between passive smoking and the risk of hypertension, and the adjusted odds ratio (OR; 95% confidence interval (CI)) was 1.38 (1.03, 1.85). Inversely, the control rate of hypertension was significantly lower in passive smokers (26.3%) than in non-passive smokers (35.7%) among the hypertensive patients; the adjusted OR (95% CI) was 0.62 (0.44, 0.87). Furthermore, a dose-response association was observed between the amount and frequency of passive cigarette smoking and the control rate of hypertension. We demonstrated that passive smoking was significantly associated with the increased prevalence and lower control rate of hypertension in a community-based elderly population in Beijing, China. Additional randomized controlled trials and large prospective studies are still required to determine the relationship between passive smoking and hypertension among the Chinese elderly.
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INTRODUCTION

Tobacco exposure remains one of the major public health challenges around the world. The 2010 Global Adult Survey reported that the prevalence of tobacco smoking was 52.9% among Chinese males and 2.4% among Chinese females.[1] As a result, many Chinese non-smokers are exposed to environmental tobacco smoke. A recently published meta-analysis reported that nearly half of the Chinese adult non-smokers were consistently exposed to environmental tobacco smoke during the past two decades.[2]

Previous evidence has demonstrated that both active and passive smoking are important risk factors for various chronic diseases, such as cancer, cardiovascular events and kidney disease.[3–6] Although active smoking is linked to hypertension,[7–9] the effect of passive smoking on hypertension has rarely been studied, and the few epidemiological studies on this topic have yielded uncertain findings.[10–12] This can be attributed to the fact that various diagnostic criteria of hypertension and different characteristics of participants contribute to the controversial relationship between passive smoking and hypertension. To the best of our knowledge, no previous study has investigated whether passive smoking is associated with the prevalence and control of hypertension in urban elderly female populations, which have a higher prevalence of hypertension and a higher risk of developing negative health outcomes.

By the year of 2050, elderly people will constitute nearly one-third of the total Chinese population.[13] However, the relationship between passive smoking and hypertension in elderly Chinese remains uncertain. Therefore, we performed a cross-sectional study to examine the association between passive smoking and the prevalence and control of hypertension among elderly non-smoking women in urban Beijing, China.

METHODS

Study sample

We used the data from a previously reported cross-sectional study.[14] We included local residents aged 60 years or above and those who lived in the Wanshoulu Community of Haidian District for more than one year. A two-stage stratified clustering sampling method was performed. First, we

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randomly selected 9 of the 94 communities in this district. Second, we randomly invited one elderly resident from each household in these 9 communities. Finally, a total of 2162 of the 2510 eligible elderly subjects (response rate: 86.1%) completed the survey from September 2009 to June 2010. After excluding men, current smokers, ever smokers and participants with incomplete data, 1,078 female non-smokers were included in the present analysis.

Data collection and measurement

A face-to-face interview was administered by trained physicians and nurses. Information on the participants' demographic characteristics, medical histories and lifestyles was recorded in a structured questionnaire. The height, weight, waist circumference and blood pressure of each participant were measured by trained nurses according to a standardized protocol. The waist circumference was measured midway between the lower rib margin and iliac crest. Height was measured in meters after participants removed their shoes. Weight was measured in kilograms after participants removed heavy clothing, and 1 kg was deducted for the remaining garments. The body mass index (BMI) was calculated as weight (in kilograms) divided by the height in meters squared. Overnight fasting blood specimens were obtained from each participant for measuring the serum lipid and glucose. Blood specimens were sent to the central laboratory of the Chinese People's Liberation Army General Hospital in fewer than 30 min.

Definitions of passive smoking status

A current smoker was defined as a person who smoked at least one cigarette or used other tobacco products at the time of the survey. An ever-smoker was defined as a person who smoked daily for at least 6 months at any point in her life.[15] As in our previous study, a passive smoker was defined as a non-smoker who was exposed to environmental smoke on at least one occasion per week, for at least 30 min per occasion.[3] Environmental smoke sources included the home, workplace and other public places. We collected information on the amount and frequency of passive cigarette smoking as continuous variables based on the following questions. (1) Did you have family members (or coworkers, or other smokers) who smoked cigarettes near you so that you frequently breathed in their smoke? (2) How many cigarettes per day were you exposed to from smokers? (3) For how long (hours) per day were you exposed to smokers? The amount of passive smoking was then categorized as follows: 0 cigarette per day, 1–10 cigarettes per day and >10 cigarettes per day. The frequency of passive cigarette smoking was categorized as follows: 0 hour per day, 1–2 hour per day and >2 hour per day.

BP measurement and definitions

In the survey, each participant's blood pressure records were obtained from 07:00–08:00 hours using standardized mercury sphygmomanometers. Although the participants were in a sitting position, trained nurses obtained blood pressure records from the right arm of each participant. Both diastolic and systolic blood pressure levels were measured twice with a short resting interval between the two measurements. The mean values of the two readings were calculated to record the blood pressure value of each participant. If the two measurements differed by more than 10 mm Hg, a third measurement was obtained from the participant, and the average value of the three records was calculated as the final record.

Hypertension was defined as systolic blood pressure $\geqslant 140$ mm Hg and/or diastolic blood pressure $\geqslant 90$ mm Hg and/or self-reported antihypertensive treatment. Among the participants defined as hypertensive patients in the present interview, the control rate of hypertension was defined as the usage of antihypertensive medication associated with systolic blood pressure <140 mm Hg and diastolic blood pressure <90 mm Hg.[16]

Statistical analysis

SPSS version 19.0 (SPSS Inc., Chicago, IL, USA) was used for data analysis. The level of statistical significance was set at a two-tailed α-value of 0.05.

Baseline characteristics were described using descriptive statistics. Differences in the means and proportions were evaluated by the *t*-test and χ^2 test. The relationships between passive smoking status (whether exposed to passive smoking and the amount and frequency of passive cigarette smoking), and diastolic and systolic blood pressure (continuous variable) were examined using a multivariable linear regression model among the 558 participants without antihypertensive treatment. Of the 1078 participants with complete baseline data, the logistic regression model was used to identify the associations between passive smoking status and the prevalence and control rate of hypertension (dichotomy variable). Participants aged less than 65 years were excluded from the sensitivity analysis. The characteristics of the study population, according to the presence or absence of passive smoking, amount of passive smoking and frequency of passive smoking are listed in the Supplementary tables.

Ethical consideration

The Independent Ethics Committee of the Chinese People's Liberation Army General Hospital (No. EC0411-2001) approved our study. Signed informed consent was obtained from each eligible subject.

RESULTS

A total of 1078 female subjects were included in the present analysis after excluding 51 current female smokers, 65 females who were ever smokers and 59 females with incomplete data. Baseline characteristics of the study population are presented in Table 1. Overall, the mean age of the female participants was 70.5 ± 6.3 years, with a range from 60 to 95 years. The average values of the daily amount and frequency of passive cigarette smoking were 3.9 cigarettes and 0.9 h, respectively. The prevalence of passive smoking was 32.1% among normotensive women. In the hypertensive patients, the prevalence of passive smoking in the uncontrolled hypertensive and controlled hypertensive patients were 41.5 and 31.4%, respectively.

Comparison of the mean systolic and diastolic blood pressure

Among participants without antihypertensive treatment ($n=558$), the comparisons of the mean values of systolic and diastolic blood pressure by passive smoking status are shown in Table 2. We observed that the mean values of systolic and diastolic blood pressure were both increased when the amount and frequency of passive cigarette smoking were elevated. Compared with non-passive smokers, participants with more than 2 h of daily passive cigarette smoking had a significantly higher systolic blood pressure (by 4.24) and higher diastolic blood pressure (by 2.09) after multiple adjustments. However, the blood pressure of the passive smokers who were exposed to 1–10 cigarettes per day or >10 cigarette per day was not significantly higher than that of the non-passive smokers.

Association between passive smoking and hypertension

Table 3 shows the unadjusted and adjusted associations between passive smoking status and the prevalence and control rate of hypertension. The prevalence of hypertension was significantly higher in passive smokers (71.9%) than that in non-passive smokers (66.1%). After adjusting for age, education level, marital status, physical activity, smoking and drinking status, BMI, family history of cardiovascular disease (CVD), treatment of diabetes and hyperlipidemia, the odds ratio (OR; 95% confidence interval (CI)) for hypertension was 1.38 (1.03, 1.85) in passive smokers compared with the reference category (no passive smoking). Furthermore, a positive association was observed between the frequency of passive cigarette smoking (>2 hours per day) and the risk of hypertension compared with non-passive smokers, and the adjusted OR (95% CI) was 1.46 (1.02, 2.09). Participants exposed to environmental smoke for 1–10 cigarettes per day had a marginally increased risk of hypertension compared with non-passive smokers, and the adjusted OR (95% CI) was 1.43 (0.99, 2.05). However, a non-significant association was shown between the amount of passive cigarette smoking

Table 1 Characteristics of the normotensive and hypertensive patients in lifelong non-smoking female participants

		Hypertensive		
Characteristics	*Normotensive (n = 343)*	*Uncontrolled (n = 499)*	*Controlled (n = 236)*	*All (n = 1078)*
Mean ± s.d.				
Age (yrs)	68.9 ± 6.3	71.6 ± 6.1	70.6 ± 6.0	70.5 ± 6.3
Height (cm)	156.0 ± 5.6	155.3 ± 5.4	155.5 ± 5.0	155.5 ± 5.4
Weight (kg)	57.7 ± 8.8	62.0 ± 9.4	60.0 ± 9.3	60.2 ± 9.4
BMI (kg m^{-2})	23.7 ± 3.2	25.7 ± 3.6	24.8 ± 3.5	24.9 ± 3.6
WC (cm)	83.2 ± 9.0	87.9 ± 8.4	85.4 ± 8.4	85.8 ± 8.8
Systolic BP (mmHg)	123.8 ± 12.3	155.7 ± 14.5	127.3 ± 10.5	139.3 ± 20.0
Diastolic BP (mmHg)	71.2 ± 7.2	82.0 ± 8.6	71.6 ± 7.8	76.3 ± 9.6
TC (mmol l^{-1})	5.5 ± 1.0	5.5 ± 1.0	5.4 ± 1.0	5.5 ± 1.0
TG (mmol l^{-1})	1.5 ± 0.7	1.9 ± 1.1	1.7 ± 0.8	1.8 ± 0.9
HDL-C (mmol l^{-1})	1.6 ± 0.4	1.4 ± 0.4	1.4 ± 0.3	1.5 ± 0.4
LDL-C (mmol l^{-1})	3.3 ± 0.8	3.4 ± 0.9	3.4 ± 0.9	3.4 ± 0.9
FPG (mmol l^{-1})	5.8 ± 1.4	6.2 ± 1.9	5.9 ± 1.2	6.0 ± 1.6
Amount of passive cigarette smoking (cigarettes per day)	3.4 ± 1.1	4.7 ± 1.3	2.9 ± 1.8	3.9 ± 1.4
Frequency of passive cigarette smoking (hours per day)	0.8 ± 0.6	1.1 ± 0.9	0.6 ± 0.4	0.9 ± 0.6
N (%)				
Educational (⩾7 yrs)	268 (78.1)	307 (61.5)	179 (75.8)	754 (69.9)
Married	282 (82.2)	394 (79.0)	190 (80.5)	866 (80.3)
Current drinker	30 (8.7)	36 (7.2)	14 (5.9)	80 (7.4)
Physical activity (⩾1 hour per day)	303 (88.3)	418 (83.8)	200 (84.7)	921 (85.4)
Passive smoking	110 (32.1)	207 (41.5)	74 (31.4)	391 (36.3)

Abbreviations: BMI, body mass index; BP, blood pressure; FPG, fasting blood-glucose; HDL-C, high density lipoprotein; LDL-C, low density lipoprotein; TC, total cholesterol; TG, triglycerides; WC, waist circumference.

Table 2 Comparison of mean systolic and diastolic BP in participants without antihypertensive treatment by passive smoking status

		Systolic BP, mmHg			*Diastolic BP, mmHg*		
	N	*Mean ± s.d.*	*β (95% CI)*[a]	P-*value*	*Mean ± s.d.*	*β (95% CI)*[a]	P-*value*
No passive smoking	359	134.8 ± 19.5	1.00		74.9 ± 8.9	1.00	
Passive smoking	199	137.6 ± 22.1	2.66 (–0.74, 6.06)	0.125	76.6 ± 10.0	1.19 (–0.40, 2.79)	0.142
Amount of passive smoking							
0 cigarette per day	359	134.8 ± 19.5	1.00		74.9 ± 8.9	1.00	
1–10 cigarettes per day	107	138.1 ± 18.9	2.74 (–1.51, 6.99)	0.206	76.3 ± 8.8	0.67 (–1.32, 2.66)	0.510
> 10 cigarettes per day	92	136.9 ± 25.4	2.57 (–1.90, 7.03)	0.259	76.9 ± 11.3	1.79 (–0.30, 3.89)	0.093
Frequency of passive smoking							
0 hour per day	359	134.8 ± 19.5	1.00		74.9 ± 8.9	1.00	
1–2 hours per day	83	135.8 ± 19.8	0.38 (–4.31, 5.07)	0.874	75.5 ± 8.8	–0.10 (–2.30, 2.10)	0.929
> 2 hours per day	116	138.9 ± 23.6	4.24 (0.16, 8.31)	0.041	77.3 ± 10.8	2.09 (0.18, 4.00)	0.032

Abbreviations: BMI, body mass index; BP, blood pressure; 95% CI, 95% confidence interval.
[a]Adjusted for age, educational level, marital status, physical activity, drinking status, BMI, family history of CVD, treatment of diabetes and hyperlipidemia.

(> 10 cigarettes per day) and risk of hypertension compared with non-passive smokers.

As presented in Table 4, in hypertensive patients ($n = 735$), the mean values of systolic and diastolic blood pressure were 148.7 and 79.9 mm Hg in passive smokers, which were significantly higher than those in non-passive smokers (systolic and diastolic blood pressure were 145.2 and 77.9 mm Hg, respectively). The control rate of hypertension was significantly lower in passive smokers (26.3%) than those in the reference category (no passive smoking, 35.7%), and the adjusted OR (95% CI) was 0.62 (0.44, 0.87). In addition, we found a significant dose-response association between the level (1–10 cigarettes per day: OR = 0.67, 95% CI 0.44, 1.02; and > 10 cigarettes per day: OR = 0.56, 95% CI 0.35, 0.89) and frequency (1–2 hours per day: OR = 0.69, 95% CI 0.43, 1.09; and > 2 hours per day: OR = 0.57, 95% CI 0.38, 0.87) of passive cigarette smoking and the control rate of hypertension. Participants exposed to environmental smoke for > 10 cigarettes per day and > 2 hours per day had a significantly increased risk of non-controlled hypertension compared with non-passive smokers.

Table 3 Association between passive smoking status and the prevalence of hypertension

	N *(%)*	*OR (95% CI)*	P-*value*	*aOR (95% CI)*[a]	P-*value*
No passive smoking	454/687 (66.1)	1.00		1.00	
Passive smoking	281/391 (71.9)	1.31 (1.00, 1.72)	0.050	1.38 (1.03, 1.85)	0.030
Amount of passive smoking					
0 cigarette per day	454/687 (66.1)	1.00		1.00	
1–10 cigarettes per day	155/214 (72.4)	1.35 (0.96, 1.89)	0.084	1.43 (0.99, 2.05)	0.055
>10 cigarettes per day	126/177 (71.2)	1.27 (0.88, 1.82)	0.198	1.33 (0.90, 1.95)	0.151
Frequency of passive smoking					
0 hours per day	454/687 (66.1)	1.00		1.00	
1–2 hours per day	117/167 (70.1)	1.20 (0.83, 1.73)	0.328	1.28 (0.86, 1.90)	0.220
>2 hours per day	164/224 (73.2)	1.40 (1.00, 1.96)	0.048	1.46 (1.02, 2.09)	0.037

Abbreviations: OR, odds ratio; 95% CI, 95% confidence interval.
[a]Adjusted for age, educational level, marital status, physical activity, drinking status, BMI, family history of CVD, treatment of diabetes and hyperlipidemia.

Table 4 Association between passive smoking status and the control rate of hypertension

	N *(%)*	*OR (95% CI)*	P-*value*	*aOR (95% CI)*[a]	P-*value*
No passive smoking	162/454 (35.7)	1.00		1.00	
Passive smoking	74/281 (26.3)	0.64 (0.46, 0.89)	0.009	0.62 (0.44, 0.87)	0.006
Amount of passive smoking					
0 cigarette per day	162/454 (35.7)	1.00		1.00	
1–10 cigarettes per day	43/155 (27.7)	0.69 (0.46, 1.03)	0.072	0.67 (0.44, 1.02)	0.061
>10 cigarettes per day	31/126 (24.6)	0.59 (0.38, 0.92)	0.020	0.56 (0.35, 0.89)	0.014
Frequency of passive smoking					
0 hour per day	162/454 (35.7)	1.00		1.00	
1–2 hours per day	33/117 (28.2)	0.71 (0.45, 1.11)	0.129	0.69 (0.43, 1.09)	0.112
>2 hours per day	41/164 (25.0)	0.60 (0.40, 0.90)	0.013	0.57 (0.38, 0.87)	0.009

Abbreviations: OR, odds ratio; 95% CI, 95% confidence interval.
[a]Adjusted for age, educational level, marital status, physical activity, drinking status, BMI, family history of CVD, treatment of diabetes and hyperlipidemia.

Sensitivity analysis

Supplementary Tables 1–3 present characteristics of the study population according to the presence or absence of passive smoking, amount of passive smoking and frequency of passive smoking. We found that the participants who were younger, had a higher BMI (higher weight and/or higher waist circumference), had higher levels of systolic and diastolic blood pressure and were current drinkers were more likely to be exposed to environmental smoke. We assessed the relationship between passive smoking status and the prevalence and control rate of hypertension in the sensitivity analysis (Supplementary Table 4). The results were similar, as shown in Table 3, after excluding participants aged 60 to 64 years ($n = 230$, 21.3%). For participants aged 65 years or above, the adjusted OR (95% CI) of having hypertension was 1.40 (0.99, 1.98), and that of controlled hypertension was 0.54 (0.36, 0.81), in female passive smokers compared with non-passive smokers.

DISCUSSION

Our study evaluated the association between passive smoking and hypertension among older non-smoking women in an urban area of Beijing. After adjusting for potential confounding variables, a positive association was observed between passive smoking and the risk of hypertension. In contrast, passive smoking status was negatively associated with controlled hypertension. Furthermore, a dose-response association was observed between the amount and frequency of passive cigarette smoking and the control rate of hypertension.

In the present study, we observed a significant trend towards increased mean values of both systolic and diastolic blood pressure in the participants who had a higher frequency of passive cigarette smoking after multiple adjustments. These findings are in agreement with several previously published studies. The Ohasama study found that passive smoking was associated with elevated home blood pressure in a Japanese female population.[12] Seyedzadeh *et al.*[17] reported that the mean systolic and diastolic blood pressure in an environmental tobacco smoke exposure group were higher than those in a non-exposure group of elementary school children. Although the categories of passive smoking and the study population differed from those in the present study, our results consistently support those findings in a community-based elderly population. However, a non-significant or marginally significant association was shown between the level of passive cigarette smoking (1–10 cigarettes per day or >10 cigarettes per day) and risk of hypertension or blood pressure levels. One possible explanation is recall bias for the number of cigarettes. Compared with the hours of exposure, it is more difficult to exactly recall the number of cigarettes passively inhaled from other smokers. In addition, the exposure time of each cigarette is not consistent because of varied smoking habits. Some smokers may throw a cigarette butt away after smoking the entire cigarette, whereas others may throw

the butt away after only a few drags. A larger sample size and accurate measurement are needed to precisely detect the association.

Previous epidemiological studies have examined the association between passive smoking status and hypertension, and some of these reports are in accordance with our findings. As in our study, Li *et al.*[11] demonstrated that passive smoking is associated with an increased risk of hypertension among non-smoking women in rural China. Their reports demonstrated that the adjusted ORs of having hypertension for passive smoking exposure levels of 1–3, 4–6 and $\geqslant 6$ occasions per week were 1.01, 2.57 and 2.59, respectively, compared with non-passive smokers. Makris *et al.*[18] also demonstrated that masked hypertension is associated with passive smoking in a dose-related manner. In the present study, we further observed that passive cigarette smoking was negatively associated with the hypertension control rate, which has not been previously reported. Furthermore, other studies have evaluated the association between active smoking and hypertension, which might help to explain or indirectly support our findings. The results of the Olivetti Heart Study confirmed that smoking cessation was associated with lower blood pressure and minor hypertension risk.[8] Oncken *et al.*[19] also confirmed that smoking cessation caused a significant reduction in systolic blood pressure. Whether non-exposure of environmental tobacco smoke helps prevent hypertension or decreases the blood pressure should be explored in further prospective cohort studies and randomized controlled studies with a larger sample size.

Beyond high blood pressure, we also found that participants who were younger, had a higher BMI, and frequently used alcohol were more likely to be passive smokers. BMI and alcohol use have also been demonstrated to be risk factors related to high blood pressure.[20,21] In fact, the main sources of passive smoking were from the participants' children.[22] Younger elderly people may still live with their unmarried children, making them more likely to be exposed to environmental smoke. We found that passive smoking was an independent risk factor for prevalent hypertension and uncontrolled hypertension after adjustment for those confounding variables.

Potential biological pathways are likely to participate in the relationship between passive smoking and the increased risk of hypertension. It is reported that active and passive smoking may confer a hypertension risk according to a similar mechanism.[11] The relationship between active smoking and hypertension has been clearly established. Smoking may increase the blood pressure via adverse effects on vasoconstriction and/or vessel dilation, inactivating baroreceptors or impairing the function of arterial walls.[23–25] Notably, the majority of cigarette smoke is released into the surrounding environment by burning tobacco.[26] Environmental smoke, also known as secondhand smoke, contains more harmful substances than the smoke inhaled by smokers themselves.[26] Secondhand smoke contains not only carbon monoxide, formaldehyde, ethanol, methane and toluene, but also a substantial amount of carcinogens, such as benzopyrene, nitrosamines, polycyclic aromatic hydrocarbons and toluene nitroso.[25,26] Previous studies have demonstrated substantial and immediate benefits of smoking cessation treatment, including improved central blood pressure, endothelial function and cardiovascular indices.[27–29] It is very likely that active and passive smokers who have inhaled these poisonous compounds have an increased risk of developing chronic diseases.

An important advantage of our study is that we included only local non-smoking female residents from a representative urban area; as a result, the subjects had homogeneous environmental factors. We excluded men because the prevalence of active smoking is high among Chinese males.[1] Therefore, we were able to solely investigate the effect of passive smoking in the present study. In addition, hypertension was diagnosed by participant certification of the disease diagnosis or direct measurement of blood pressure. Although the present study had a cross-sectional design, our data on hypertension should not have been affected by reporting bias. Finally, because the prevalence of active smoking in Chinese females was $<3\%$,[1] the possibility of misclassification of current or ever smokers as never smokers should be very low.

Admittedly, our study has several limitations. First, we did not collect information on dietary factors, such as detailed consumption of sodium, potassium, calcium and protein. These factors might have affected the association between passive smoking and hypertension. Second, although we adjusted the potential confounding variables in the multiple regression model, other undetected covariates, such as socio-economic status, might have affected our results. For example, subjects with a low social status and/or low income are more likely to be passive smokers. In addition, these subjects may not have a sufficient economic condition for measuring and controlling their blood pressure. Third, information on the passive smoking status relied on self-reports; as a result, recall bias and reporting bias cannot be avoided. For instance, participants who were exposed to the same number of cigarettes or had the same number of hours of smoke exposure may inhale different concentrations of environmental smoke because of different ventilation conditions. A biological marker of passive smoking exposure, such as the cotinine level, is required to accurately detect the association in the future. Another limitation is that we did not categorize different sources of environmental tobacco smoke exposure because of the small sample size in the present study. High-quality randomized controlled trials and long-term follow-up cohorts are still needed to address this issue.

In conclusion, we demonstrated that passive smoking was significantly associated with an increased prevalence and lower control rate of hypertension after adjusting for potential confounding variables in a community-based elderly population from Beijing, China. Further randomized controlled trials and prospective studies are required to determine the relationship between passive smoking and hypertension among the Chinese elderly.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Author contributions: YH and BJ helped design the study and analyzed the data. BJ, DLS and JHW helped with the data collection and field operations. All of the authors helped prepare the manuscript.

1 Yang GH, Li Q, Wang CX, Hsia J, Yang Y, Xiao L, Yang J, Zhao LH, Zhang J, Xie L. Findings from 2010 Global Adult Tobacco Survey: implementation of MPOWER policy in China. *Biomed Environ Sci* 2010; **23**: 422–429.
2 Zeng J, Yang S, Wu L, Wang J, Wang Y, Liu M, Zhang D, Jiang B, He Y. Prevalence of passive smoking in the community population aged 15 years and older in China: a systematic review and meta-analysis. *BMJ Open* 2016; **6**: e009847.
3 He Y, Lam TH, Jiang B, Wang J, Sai X, Fan L, Li X, Qin Y, Hu FB. Passive smoking and risk of peripheral arterial disease and ischemic stroke in Chinese women who never smoked. *Circulation* 2008; **118**: 1535–1540.
4 Glymour MM, DeFries TB, Kawachi I, Avendano M. Spousal smoking and incidence of first stroke: the Health and Retirement Study. *Am J Prev Med* 2008; **35**: 245–248.
5 Flouris AD, Vardavas CI, Metsios GS, Tsatsakis AM, Koutedakis Y. Biological evidence for the acute health effects of secondhand smoke exposure. *Am J Physiol Lung Cell Mol Physiol* 2010; **298**: L3–L12.

6 Bastian LA, Gray KE, DeRycke E, Mirza S, Gierisch JM, Haskell SG, Magruder KM, Wakelee HA, Wang A, Ho GY, LaCroix AZ. Differences in active and passive smoking exposures and lung cancer incidence between veterans and non-veterans in the women's health initiative. *Gerontologist* 2016; **56** (Suppl 1): S102–S111.
7 Dochi M, Sakata K, Oishi M, Tanaka K, Kobayashi E, Suwazono Y. Smoking as an independent risk factor for hypertension: a 14-year longitudinal study in male Japanese workers. *Tohoku J Exp Med* 2009; **217**: 37–43.
8 D'Elia L, De Palma D, Rossi G, Strazzullo V, Russo O, Iacone R, Fazio V, Strazzullo P, Galletti F. Not smoking is associated with lower risk of hypertension: results of the Olivetti Heart Study. *Eur J Public Health* 2014; **24**: 226–230.
9 Thuy AB, Blizzard L, Schmidt MD, Luc PH, Granger RH, Dwyer T. The association between smoking and hypertension in a population-based sample of Vietnamese men. *J Hypertens* 2010; **28**: 245–250.
10 Sipetic Grujicic S, Terzic Supic Z, Nikolic Z, Gredic D, Bjekic M, Bjegovic V, Ratkov I. Risk factors for the development of arterial hypertension. *Med Glas (Zenica)* 2014; **11**: 19–25.
11 Li N, Li Z, Chen S, Yang N, Ren A, Ye R. Effects of passive smoking on hypertension in rural Chinese nonsmoking women. *J Hypertens* 2015; **33**: 2210–2214.
12 Seki M, Inoue R, Ohkubo T, Kikuya M, Hara A, Metoki H, Hirose T, Tsubota-Utsugi M, Asayama K, Kanno A, Obara T, Hoshi H, Totsune K, Satoh H, Imai Y. Association of environmental tobacco smoke exposure with elevated home blood pressure in Japanese women: the Ohasama study. *J Hypertens* 2010; **28**: 1814–1820.
13 Population division of the department of economic and social affairs of the United Nations secretariat, World Population Prospects The 2010 revision [on-line]. Available at http://esa.un.org/unpd/wpp/unpp/panel_population.htm Accessed 10 March 2013.
14 Wu L, He Y, Jiang B, Liu M, Wang J, Yang S, Wang Y. The association between the prevalence, treatment and control of hypertension and the risk of mild cognitive impairment in an elderly urban population in China. *Hypertens Res* 2016; **39**: 367–375.
15 Organization WH. *Guidelines for the conduct of tobacco-smoking surveys among health professionals: report of a WHO meeting held in Winnipeg, Canada, 7-9 July 1983 in collaboration with UICC and ACS*. World Health Organization: Geneva, 1984.
16 Gu D, Reynolds K, Wu X, Chen J, Duan X, Muntner P, Huang G, Reynolds RF, Su S, Whelton PK, He J InterASIA Collaborative Group. The International Collaborative Study of Cardiovascular Disease in ASIA. Prevalence, awareness, treatment, and control of hypertension in china. *Hypertension* 2002; **40**: 920–927.
17 Seyedzadeh A, Hashemi F, Soleimani A. Relationship between blood pressure and passive smoking in elementary school children. *Iran J Pediatr* 2012; **22**: 351–356.
18 Makris TK, Thomopoulos C, Papadopoulos DP, Bratsas A, Papazachou O, Massias S, Michalopoulou E, Tsioufis C, Stefanadis C. Association of passive smoking with masked hypertension in clinically normotensive nonsmokers. *Am J Hypertens* 2009; **22**: 853–859.
19 Oncken CA, White WB, Cooney JL, Van Kirk JR, Ahluwalia JS, Giacco S. Impact of smoking cessation on ambulatory blood pressure and heart rate in postmenopausal women. *Am J Hypertens* 2001; **14**: 942–949.
20 Hong SW, Linton JA, Shim JY, Lee HR, Kang HT. Association of alcohol consumption pattern with risk of hypertension in Korean adults based on the 2010-2012 KNHANES. *Alcohol* 2016; **54**: 17–22.
21 Feng RN, Zhao C, Wang C, Niu YC, Li K, Guo FC, Li ST, Sun CH, Li Y. BMI is strongly associated with hypertension, and waist circumference is strongly associated with type 2 diabetes and dyslipidemia, in northern Chinese adults. *J Epidemiol* 2012; **22**: 317–323.
22 Yang S, He Y, Liu M, Wang Y, Wu L, Wang J, Zhang D, Zeng J, Jiang B, Li X. Changes in and patterns of smoking exposure in an elderly urban population in Beijing: 2001-2010. *PLoS ONE* 2015; **10**: e0118500.
23 Ball K, Turner R. Smoking and the heart: the basis for action. *Lancet* 1974; **2**: 822–826.
24 Centers for Disease Control and Prevention (US); National Center for Chronic Disease Prevention and Health Promotion (US); Office on Smoking and Health (US). *How tobacco smoke causes disease: the biology and behavioral basis for smoking-attributable disease: a report of the surgeon general*. Centers for Disease Control and Prevention (US): Atlanta (GA, 2010.
25 Suarez-Varela MM, Llopis-González A, González Albert V, López-Izquierdo R, González-Manzano I, Cháves J, Biosca VH, Martin-Escudero JC. Zinc and smoking habits in the setting of hypertension in a Spanish populations. *Hypertens Res* 2015; **38**: 149–154.
26 Fielding JE, Phenow KJ. Health effects of involuntary smoking. *N Engl J Med* 1988; **319**: 1452–1460.
27 Taylor BA, Zaleski AL, Dornelas EA, Thompson PD. The impact of tetrahydrobiopterin administration on endothelial function before and after smoking cessation in chronic smokers. *Hypertens Res* 2016; **39**: 144–150.
28 Minami J, Ishimitsu T, Matsuoka H. Effects of smoking cessation on blood pressure and heart rate variability in habitual smokers. *Hypertension* 1999; **33** (1 Pt 2): 586–590.
29 Takami T, Saito Y. Effects of smoking cessation on central blood pressure and arterial stiffness. *Vasc Health Risk Manag* 2011; **7**: 633–638.

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