Association of Blood Pressure With Stroke Risk in Chinese Population: A 6-Year Prospective Cohort Study

Qi Yu
the First Hospital of Putian
Qing-Dong Jin (jqd2007@126.com)
the First Hospital of Putian
Yan-Qing Chen
the First Hospital of Putian
Xin-Ru Liu
the First Hospital of Putian
Qing-Ying Lin
the First Hospital of Putian

Research Article

Keywords: Stroke, Blood pressure, Epidemiology

Posted Date: June 9th, 2021

DOI: https://doi.org/10.21203/rs.3.rs-583220/v1

Abstract

Objective: Previous results on the association between blood pressure(BP) and stroke risk were controversial. We investigated the association of BP with stroke risk in China.

Method: We included a total of 5,700 adults aged 40-90 years from the China Health and Nutrition Survey (CHNS) cohort. Cox proportional hazards regression models were used to estimate hazard ratios and 95% CIs. Restricted cubic spline analyses were used to explore linear and nonlinear relationships of BP and stroke.

Result: With a median follow-up of 6 years, a total of 5,700 individuals were enrolled in our study, of whom 178 developed stroke. Multivariable adjusted Cox models including systolic blood pressure(SBP) and diastolic blood pressure(DBP) showed a strong positive association between SBP and overall stroke. Compared with participants with SBP 0-130 mmHg, the multivariable adjusted HRs[95% CIs] in participants with SBP 130-140, 140-160, 160-180, and ≥180 mmHg were 1.08[0.62-1.89], 2.41[1.51-3.86], 2.21[1.16-4.20], and 3.90[1.78-8.55] for overall stroke; 0.65[0.21-2.04], 3.68[1.73-7.83], 2.51[0.84-7.47], and 5.91[1.69-20.60] for ischemic stroke; 1.26[0.50-3.20], 1.19[0.47-3.04], 2.06[0.66-6.41], and 5.10[1.36-19.20] for hemorrhagic stroke. Restricted cubic spline analyses including SBP and DBP showed linear relationships of SBP with overall, ischemic and hemorrhagic stroke. No linear or nonlinear relationships of DBP with overall, ischemic and hemorrhagic stroke were observed.

Conclusion: SBP is independently and directly related to the risk of overall and its subtypes. Besides, the risk of ischemic and hemorrhagic stroke might be higher when SBP were more than 140 mmHg and 160 mmHg.

1. Introduction

Stroke is the leading cause of death and permanent disability worldwide[1][2][3]. In China, the most populous country in the world, the incidence of stroke among adults over the age of 40 years had increased by 8.3% from 2002 to 2013[4][5]. Hypertension is the most important modifiable risk factor for stroke, with a strong, direct, linear, and continuous relationship between blood pressure(BP) and cardiovascular disease (CVD) risk[6][7]. Specifically, a meta-analysis of 61 cohorts recruited between 1950 and 1990 reported that usual blood pressure is strongly and directly related to vascular (and overall) mortality, without any evidence of a threshold down to at least 115/75 mm Hg, in participants aged 40–89 years[7].

In China, the prevalence of hypertension rapidly increased in the past 30 years. However, awareness, treatment and control of hypertension had declined or remained unchanged in China from 2000 to 2010[5][8], while they had increased significantly in the developed countries[9], these may affect the incidence of stroke. Despite the high burden of stroke in China, the association between BP and stroke has not been fully investigated by large prospective studies on Chinese. To better understand the etiology and prevent stroke, more evidence on the association between BP and stroke is needed. Thus, we

investigated the association of BP levels with overall stroke and its subtypes among 5,700 adults from nine provinces around China.

2. Method

2.1. Study design

The China Health and Nutrition Survey(CHNS) was conducted by the University of North Carolina at Chapel Hill, and the National Institute for Nutrition and Health at the Chinese Center for Disease Control and Prevention to examine the status of economic, public resources, health and nutrition. All methods were performed in accordance with the relevant guidelines and regulations. Details about the study design were available elsewhere[10][11]. A multistage, random cluster process was used to draw the samples in the Chinese population. The survey was an ongoing nationwide study that started in 1989 and subsequently conducted in 1991, 1993, 1997, 2000, 2004, 2006, 2009, 2011 and 2015 in nine provinces(Heilongjiang, Liaoning, Shandong, Jiangsu, Henan, Hubei, Hunan, Guangxi, and Guizhou) of China. Baseline data collection included demographic information, medical history, standardized medical examination, laboratory tests, and anthropometric measurements.

2.2. Study population

For this study, data were drawn from the 2009–2015 CHNS cycles. We included Chinese residents with BP data collected at age 40 to 90 years and with subsequent follow-up time available(n = 7,717). After excluding persons who were followed up only until 2011 and did not participate in the later follow-up survey in 2015(n = 958) and those without complete BP measure data at baseline(n = 1,059). As a result, 2,680 men and 3,020 women were available for analysis.

2.3. Exposure,covariates and outcomes

Adults and children received detailed physical examinations that included weight, height, arm and head circumference, mid-arm skinfold measurements, and BP. BP was measured thrice by experienced physicians with the participant in the sitting position. Systolic blood pressure(SBP) and diastolic blood pressure(DBP) were defined as mean SBP and DBP of three test results.

History of diseases, individual activities, lifestyle, health status, marriage and birth history were acquired through an individual questionnaire. Smoking status was classified into two categories as follows: current smoker or not. Drinking status was divided into two groups: current drinker or not. Diabetes was identified by self-reports of a history of diabetes diagnosis. The biomarker data collected in CHNS 2009 involves the release of 26 fasting blood measures on individuals aged 7 and older. Frozen serum samples were sent to a national central lab in Beijing for measurement of serum lipid levels.

Incident stroke was diagnosed by a doctor’s diagnosis or treatment history for stroke during the follow-up period(2009–2015). Cases were censored at the date of diagnosis of stroke or the final visit, whichever came first.

2.4. Statistical analysis

Results are presented as mean ± standard deviation or median (interquartile range) for continuous variables, and number (percentage) for categorical variables. Mantel-Haenszel $\chi 2$ test was used for categorical variables. T-test was used for continuous variables which were normally distributed and Wilcoxon rank-sum test was used for variables which were not normally distributed.

We used cox proportional hazards models to explore the relation between BP level and overall stroke incidence[12]. In addition, we have also established models with outcomes of ischemic stroke and hemorrhagic stroke(excluding unknown stroke participants). Fully adjusted models were adjusted for potential confounders including age, gender(male or female), body mass index(BMI, kg/m^2), total cholesterol(TC, mmol/L), drinking status(current drinker or not), smoking status (current smoker or not), diabetes(no or yes), CVD history, and site(rural or urban). In addition, to examine the independent association of SBP and DBP with stroke risk, we further ran a multivariable model including both SBP and DBP. Tests for linear trend were computed by modeling the median values of each category as a continuous variable in regression models.

Restricted cubic spline analyses with 4 knots (5th, 35th, 65th and 95th) were also used to explore the potential linear or nonlinear relationship of BP level with overall stroke, ischemic and hemorrhagic stroke. The interaction was tested by modeling the interaction between SBP and sex, age and BMI for stroke risk. For a sensitivity analysis, we additionally examined the shape of BP-stroke risk relation after winsorization of SBP and DBP at the 1st/99th.

All statistical analyses were carried out using R software version 3.6.1 (http://www.R-project.org). All tests were 2 sided, and $P < 0.05$ was considered statistically significant.

3. Result

Baseline characteristics of 5,700 study participants are presented in Table 1. During the follow-up, 178 participants developed stroke events, including 65 ischemic strokes, 51 hemorrhagic strokes, and 62 unspecified stroke events.

Table 1
Baseline clinical characteristics of participants

Characteristics	Total	Gender		
		Female	Male	P
Age(year)	56.00(48.00–65.00)	56.00(48.00–65.00)	56.50(48.00–65.00)	0.568
Gender				
female	3020(52.98%)	NA	NA	
male	2680(47.02%)	NA	NA	
BMI(kg/m^2)	23.38(21.21–25.76)	23.47(21.34–25.97)	23.28(21.06–25.50)	0.001
SBP(mmHg)	125.00(116.00–139.00)	123.00(113.00–139.00)	126.00(118.00–139.00)	< .001
DBP(mmHg)	80.00(74.00–89.00)	80.00(72.00–87.00)	81.00(77.00–90.00)	< .001
TC(mmol/L)	4.91(4.32–5.62)	5.00(4.39–5.73)	4.82(4.24–5.49)	< .001
Current smoker				
no	4079(71.60%)	2919(96.69%)	1160(43.32%)	< .001
yes	1618(28.40%)	100(3.31%)	1518(56.68%)	
Current drinker				
no	3830(67.20%)	2762(91.49%)	1068(39.85%)	< .001
yes	1869(32.80%)	257(8.51%)	1612(60.15%)	
Diabetes				
no	5484(96.45%)	2915(96.84%)	2569(96.00%)	0.087
yes	202(3.55%)	95(3.16%)	107(4.00%)	
CVD history				
no	5558(97.54%)	2960(98.08%)	2598(96.94%)	0.006
yes	140(2.46%)	58(1.92%)	82(3.06%)	
Site				
urban	1758(30.84%)	935(30.96%)	823(30.71%)	0.838
rural	3942(69.16%)	2085(69.04%)	1857(69.29%)	
Stroke type				
Ischemic stroke	65(36.52%)	24(35.82%)	41(36.94%)	0.043

Characteristics	Total	Gender		
		Female	Male	P
Hemorrhagic stroke	51(28.65%)	13(19.40%)	38(34.23%)	
Unknown	62(34.83%)	30(44.78%)	32(28.83%)	

3.1. Association of BP level with overall stroke

Table 2 displays the association of BP level with overall stroke. In multivariable adjusted models(model 1 and model 2), SBP and DBP showed a linear association with overall stroke, respectively. However, in a mutually adjusted model(model 3) including both SBP and DBP, the association with the risk of overall stroke was positive for SBP, but not significant for DBP. Compared with participants with SBP 0-130 mmHg, the multivariable adjusted HRs[95% CIs] (model 3) were 1.08[0.62–1.89], 2.41[1.51–3.86], 2.21[1.16–4.20], and 3.90[1.78–8.55] for overall stroke in participants with SBP 130–140, 140–160, 160–180, and ≥ 180 mmHg, respectively; a significant positive trend in categorized SBP for incident overall stroke (P for trend < 0.001) was observed; each additional 1 mmHg of SBP was associated with a 2% higher probability of developing overall stroke(1.02[1.01–1.03]).

Multivariable adjusted restricted cubic spline analyses (model 3)showed a linear association of SBP with overall stroke (P for linearity < 0.001); no significant linear and nonlinear relationships of DBP with overall stroke were observed(Fig. 1). Multivariable adjusted restricted cubic spline analyses (model 1 and model 2) showed linear association of SBP and DBP with overall stroke, respectively (Figure s1 and Figure s2 in appendix).

We found no significant age, gender and BMI interaction for the association of SBP with overall stroke (P interaction = 0.48, 0.902, and 0.764 for age, gender and BMI with overall stroke)(Fig. 2).

Table 2
Associations of BP with overall stroke

	Model 1		Model 2		Model 3	
	HR[95%CI]	P	HR[95%CI]	P	HR[95%CI]	P
SBP(mmHg)						
0-130	1.00		1.00		1.00	
130–140	1.41[0.84–2.35]	0.194	1.19[0.69–2.05]	0.542	1.08[0.62–1.89]	0.785
140–160	3.85[2.62–5.66]	< .001	2.86[1.87–4.36]	< .001	2.41[1.51–3.86]	< .001
160–180	4.35[2.64–7.17]	< .001	2.85[1.63–4.98]	< .001	2.21[1.16–4.20]	0.016
≥ 180	7.69[4.16–14.20]	< .001	5.62[2.96–10.66]	< .001	3.90[1.78–8.55]	< .001
P for trend		< .001		< .001		< .001
P for one unit increase	1.03[1.02–1.04]	< .001	1.03[1.02–1.03]	< .001	1.02[1.01–1.03]	< .001
DBP(mmHg)						
0–80	1.00		1.00		1.00	
80–90	1.68[1.11–2.53]	0.013	1.89[1.21–2.95]	0.005	1.50[0.95–2.37]	0.081
90–100	2.78[1.81–4.27]	< .001	2.82[1.76–4.50]	< .001	1.84[1.11–3.06]	0.018
100–110	3.26[1.87–5.67]	< .001	2.94[1.63–5.31]	< .001	1.48[0.76–2.87]	0.252
≥ 110	6.58[3.55–12.19]	< .001	5.35[2.56–11.19]	< .001	1.93[0.81–4.60]	0.138
P for trend		< .001		< .001		0.092
P for one unit increase	1.04[1.03–1.05]	< .001	1.03[1.02–1.05]	< .001	1.01[0.99–1.03]	0.230

Model 1 adjusted for age and gender. Model 2 adjusted for age, gender, BMI, TC, smoking status, drinking status, diabetes, CVD history and site. Model 3 based on model 2, mutually adjusted for SBP and DBP.

Hazard ratios are indicated by solid lines and 95% CI by shaded areas. A reference point is the median value for each SBP and DBP, with knots placed at 5th, 35th, 65th, and 95th centiles of each SBP and DBP. All models were adjusted for age, gender, BMI, TC, smoking status, drinking status, diabetes, CVD history, site and mutually adjusted for SBP and DBP.

Hazard ratios are indicated by solid lines. A reference point is the median value for each SBP and DBP, with knots placed at 5th, 35th, 65th, and 95th centiles of each SBP and DBP. All models were adjusted for age, gender, BMI, TC, smoking status, drinking status, diabetes, CVD history, site and mutually adjusted for SBP and DBP.

3.2. Association of BP level with ischemic stroke

Table 3 displays the association of BP level with ischemic stroke. In multivariable adjusted models(model 1 and model 2), SBP and DBP showed a linear association with ischemic stroke, respectively. However, in a mutually adjusted model(model 3) including both SBP and DBP, the association with the risk of ischemic stroke was positive for SBP, but not significant for DBP. Compared with participants with SBP 0-130 mmHg, the multivariable adjusted HRs[95% CIs] (model 3) were 0.65[0.21–2.04], 3.68[1.73–7.83], 2.51[0.84–7.47], and 5.91[1.69–20.60] for ischemic stroke in participants with SBP 130–140, 140–160, 160–180, and ≥ 180 mmHg, respectively; a significant positive trend in categorized SBP for incident ischemic stroke (P for trend 0.002) was observed; each additional 1 mmHg of SBP was associated with a 3% higher probability of developing ischemic stroke(1.03[1.01–1.04]) (Table 3).

Multivariable adjusted restricted cubic spline analyses (model 3) showed a linear association of SBP with ischemic stroke (P for linearity < 0.001); no significant linear and nonlinear relationships of DBP with ischemic stroke were observed(Fig. 1). Multivariable adjusted restricted cubic spline analyses (model 1 and model 2) showed linear association of SBP and DBP with ischemic stroke, respectively (Figure s1 and Figure s2 in appendix).

We found no significant age, gender and BMI interaction for the association of SBP with ischemic stroke (P interaction = 0.417, 0.664, and 0.626 for age, gender and BMI with ischemic stroke)(Fig. 2).

Table 3
Associations of BP with ischemic stroke

	Model 1		Model 2		Model 3	
	HR[95%CI]	P	HR[95%CI]	P	HR[95%CI]	P
SBP(mmHg)						
0-130	1.00		1.00		1.00	
130–140	0.68[0.23–2.03]	0.486	0.69[0.22–2.15]	0.527	0.65[0.21–2.04]	0.458
140–160	4.55[2.47–8.38]	< .001	4.16[2.11–8.20]	< .001	3.68[1.73–7.83]	< .001
160–180	3.14[1.27–7.78]	0.013	3.02[1.15–7.91]	0.025	2.51[0.84–7.47]	0.099
≥ 180	8.94[3.44–23.20]	< .001	7.71[2.81–21.15]	< .001	5.91[1.69–20.60]	0.005
P for trend		< .001		< .001		0.002
P for one unit increase	1.03[1.02–1.04]	< .001	1.03[1.02–1.04]	< .001	1.03[1.01–1.04]	< .001
DBP(mmHg)						
0–80	1.00		1.00		1.00	
80–90	1.17[0.60–2.30]	0.644	1.21[0.59–2.47]	0.606	0.93[0.45–1.93]	0.842
90–100	2.59[1.33–5.04]	0.005	2.64[1.31–5.33]	0.007	1.60[0.74–3.45]	0.231
100–110	3.19[1.37–7.47]	0.007	2.92[1.19–7.15]	0.019	1.28[0.46–3.57]	0.635
≥ 110	4.43[1.48–13.31]	0.008	5.47[1.73–17.34]	0.004	1.51[0.38–6.08]	0.560
P for trend		< .001		< .001		0.345
P for one unit increase	1.04[1.02–1.06]	< .001	1.04[1.02–1.06]	< .001	1.01[0.98–1.04]	0.532

Model 1 adjusted for age and gender. Model 2 adjusted for age, gender, BMI, TC, smoking status, drinking status, diabetes, CVD history and site. Model 3 based on model 2, mutually adjusted for SBP and DBP.

3.3. Association of BP level with hemorrhagic stroke

Table 4 displays the association of BP level with hemorrhagic stroke. In multivariable adjusted models(model 1 and model 2), SBP and DBP showed a linear association with hemorrhagic stroke,

respectively. However, in a mutually adjusted model(model 3) including both SBP and DBP, the association with the risk of hemorrhagic stroke was positive for SBP, but not significant for DBP. Compared with participants with SBP 0-130 mmHg, the multivariable adjusted HRs[95% CIs] (model 3) were 1.26[0.50–3.20], 1.19[0.47–3.04], 2.06[0.66–6.41], and 5.10[1.36–19.20] for hemorrhagic stroke in participants with SBP 130–140, 140–160, 160–180, and ≥ 180 mmHg, respectively; a significant positive trend in categorized SBP for incident hemorrhagic stroke (P for trend 0.032) was observed; each additional 1 mmHg of SBP was associated with a 2% higher probability of developing hemorrhagic stroke(1.02[1.00-1.04]) (Table 4).

Multivariable adjusted restricted cubic spline analyses (model 3) showed a linear association of SBP with hemorrhagic stroke (P for linearity 0.022); no significant linear and nonlinear relationships of DBP with hemorrhagic stroke were observed(Fig. 1). Multivariable adjusted restricted cubic spline analyses (model 1 and model 2) showed linear association of SBP and DBP with hemorrhagic stroke, respectively (Figure s1 and Figure s2 in appendix).

We found no significant age, gender and BMI interaction for the association of SBP with hemorrhagic stroke (P interaction = 0.532, 0.519, and 0.605 for age, gender and BMI with hemorrhagic stroke)(Fig. 2).

Table 4
Associations of BP with hemorrhagic stroke

	Model 1		Model 2		Model 3	
	HR[95%CI]	P	HR[95%CI]	P	HR[95%CI]	P
SBP(mmHg)						
0-130	1.00		1.00		1.00	
130–140	1.98[0.87–4.49]	0.104	1.33[0.53–3.29]	0.543	1.26[0.50–3.20]	0.626
140–160	2.01[0.91–4.41]	0.083	1.30[0.55–3.09]	0.548	1.19[0.47–3.04]	0.710
160–180	4.72[1.98–11.23]	< .001	2.34[0.86–6.35]	0.094	2.06[0.66–6.41]	0.214
≥ 180	10.22[3.82–27.32]	< .001	6.22[2.21–17.51]	< .001	5.10[1.36–19.20]	0.016
P for trend		< .001		0.002		0.032
P for one unit increase	1.03[1.02–1.04]	< .001	1.02[1.01–1.04]	< .001	1.02[1.00-1.04]	0.019
DBP(mmHg)						
0–80	1.00		1.00		1.00	
80–90	1.99[0.96–4.14]	0.064	2.84[1.19–6.76]	0.019	2.27[0.93–5.51]	0.071
90–100	1.50[0.60–3.74]	0.383	1.76[0.63–4.91]	0.284	1.22[0.42–3.59]	0.716
100–110	3.95[1.53–10.18]	0.005	4.16[1.41–12.24]	0.010	2.09[0.61–7.15]	0.240
≥ 110	5.94[1.89–18.72]	0.002	3.94[0.78–19.79]	0.096	1.37[0.22–8.70]	0.738
P for trend		0.001		0.025		0.694
P for one unit increase	1.04[1.01–1.06]	< .001	1.03[1.00-1.05]	0.023	1.01[0.97–1.04]	0.744

Model 1 adjusted for age and gender. Model 2 adjusted for age, gender, BMI, TC, smoking status, drinking status, diabetes, CVD history and site. Model 3 based on model 2, mutually adjusted for SBP and DBP.

4. Discussion

We used data from the CHNS study, which was a nationally representative prospective cohort in nine provinces around China, enabling us to provide a general method to estimate the association of BP and risk of stroke for the Chinese population. In this study, increasing SBP was an independent risk factor and positively associated with overall stroke, ischemic stroke, and hemorrhagic stroke. The risks of ischemic and hemorrhagic stroke were higher when SBP was more than 140 mmHg and 160 mmHg(Table 2, Table 3 and Table 4). Multivariable adjusted restricted cubic spline analyses showed linear association of SBP with overall, ischemic and hemorrhagic stroke ($P < 0.001$, $P < 0.001$ and $P = 0.022$).

A previous study proved that rising SBP and DBP levels were associated with the development of CVD[13]. Higher SBP level was positively associated with increased risk of CVD with adjustment or stratification for DBP[14]. By contrast, another study demonstrated that DBP was not consistently associated with the risk of CVD, after adjusting or stratification for SBP [15][16]. Similar to previous results, our findings showed that SBP and DBP were positively associated with stroke in the multivariate model(model 1 and model 2). However, in a mutually adjusted model(model 3) including both SBP and DBP, the association with the risk of overall stroke was positive for SBP, but not significant for DBP(Table 2, Table 3 and Table 4).

In our study, the relation of SBP to the risk of overall stroke is continuous and graded and no critical value has been observed(Table 2, Fig. 1). Consistently, in another retrospective study with 1.25 million patients[17], a dose-response association for SBP with overall stroke had also been observed. Further, in a low-income population in China, significant linear associations of SBP to the risk of overall stroke had also been reported in a prospective cohort study[18]. In addition, a meta-analysis involved nearly 1 million participants and 56,000 vascular deaths from 61 prospective studies has also confirmed the continued and graded impact of BP on CVD risk[7]. This prospective research observed that BP was associated with vascular mortality, without any clear indication of a threshold. In the entire BP range, every 20 mmHg increase in SBP (or 10 mm Hg increase in DBP) will double the risk of stroke or coronary heart disease for people between 40 and 69 years of age. Furthermore, findings from the Framingham study further suggested that there is a continuous, graded association of SBP to the rate of development of CVD at all ages, without any indication of a threshold was observed in any group from age 35 to 84 years[19]. Other studies have also reported similar linear associations of BP to coronary heart disease (CHD) and all-cause mortality [20][21].

In this study, compared to the reference group (SBP < 130 mmHg), the ischemic and hemorrhagic stroke risks were significantly increased among those with SBP values ≥ 140 mmHg and ≥ 160 mmHg, after adjusting other conventional CVD risk factors(model 2 in Table 3 and Table 4). The ideal BP cut-off values for decreasing stroke risk were SBP values ≤ 140 mmHg and ≤ 160 mmHg for ischemic stroke and hemorrhagic stroke. These results were similar to a prospective cohort study[18], showing that stroke risk significantly increased among individuals with SBP levels ≥ 140 mmHg (for ischemic stroke) and ≥ 160 mmHg (for hemorrhagic stroke), compared with those with SBP levels < 130 mmHg. Further, in another retrospective study with 1.25 million patients[17], for SBP levels ≥ 160 mmHg, SBP was more

strongly associated with hemorrhagic stroke than ischaemic stroke. The stroke subtypes for the association of BP and stroke risk needs to be demonstrated in further studies.

We found no significant age, sex and BMI interaction for the associations of SBP with ischemic stroke, hemorrhagic stroke, and overall stroke(Fig. 2). In sensitivity analysis, the shape of BP-stroke risk relation after winsorization of SBP and DBP at the 1st/99th did not change substantially (Figure s3 in appendix).

Our study has several limitations. First, we only screen residents older than 40 and younger than 90 years old from nine provinces of China. Therefore, our current results cannot be generalized to all populations in China. Second, the long-term, > 6 years, incidence of stroke for BP is unknown. In future studies, we project to prospective follow-up participants, gather data about mortality and incidence of stroke in participants. Finally, we did not collect information on usage of BP-lowering medication or treatment at baseline for participants from CHNS data.

5. Conclusion

In conclusion, our study found the relationship between BP and stroke subtypes in the Chinese general population. SBP is strongly and directly related to the risk of overall stroke, without any evidence of a threshold. Besides, the risk of ischemic, and hemorrhagic stroke might be higher when SBP were more than 140 mmHg and 160 mmHg. These results provide more evidence for guidelines or health policies of primary prevention of stroke subtypes with the management of BP level. Individual-level and population-level interventions to control BP level are needed to reduce the burden of stroke in China.

List Of Abbreviations

BMI	body mass index
BP	blood pressure
CHD	coronary heart disease
CHNS	China Health and Nutrition Survey
CVD	cardiovascular disease
DBP	diastolic blood pressure
HDL-C	high-density lipoprotein cholesterol
LDL-C	low-density lipoprotein cholesterol
SBP	systolic blood pressure
TC	total cholesterol

Declarations

Ethics approval and consent to participate

This research uses data from the China Health and Nutrition Survey (CHNS). The institutional review committees from the University of North Carolina at Chapel Hill and the National Institute for Nutrition and Food Safety, China Centre for Disease Control and Prevention, approved the survey protocols and instruments and the process for obtaining informed consent for the survey. All participants and/or their parents/guardians provided written informed consents for their participation in the survey.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and/or analysed during the current study are available in the CHNS repository, http://www.cpc.unc.edu/projects/china/data/datasets.

Competing interests

The authors declare that there is no conflict of interest.

Funding

Not applicable.

Authors' contributions

QY and QDJ conceptualized and designed the study, carried out the initial analyses, drafted the initial manuscript, and reviewed and revised the manuscript; YQC, QYL and XRL critically reviewed and revised the manuscript; and all authors approved the final manuscript for submission.

Acknowledgements

We acknowledge CHNS database for providing their platforms and contributors for uploading their meaningful datasets.

References

1. Feigin, Valery L et al. "Global Burden of Stroke." Circulation research vol. 120,3 (2017): 439–448. doi:10.1161/CIRCRESAHA.116.308413
2. Writing Group Members et al. "Heart Disease and Stroke Statistics-2016 Update: A Report From the American Heart Association." Circulation vol. 133,4 (2016): e38-360. doi:10.1161/CIR.0000000000000350

3. Feigin, Valery L et al. “Global and regional burden of stroke during 1990–2010: findings from the Global Burden of Disease Study 2010.” Lancet (London, England) vol. 383,9913 (2014): 245 – 54. doi:10.1016/s0140-6736(13)61953-4
4. Guan, Tianjia et al. “Rapid transitions in the epidemiology of stroke and its risk factors in China from 2002 to 2013.” Neurology vol. 89,1 (2017): 53–61. doi:10.1212/WNL.0000000000004056
5. Wang, Zhenkun et al. “Age-Period-Cohort Analysis of Stroke Mortality in China: Data From the Global Burden of Disease Study 2013.” Stroke vol. 48,2 (2017): 271–275. doi:10.1161/STROKEAHA.116.015031
6. Chobanian, Aram V et al. “Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure.” Hypertension (Dallas, Tex.: 1979) vol. 42,6 (2003): 1206–52. doi:10.1161/01.HYP.0000107251.49515.c2
7. Lewington, Sarah et al. “Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies.” Lancet (London, England) vol. 360,9349 (2002): 1903-13. doi:10.1016/s0140-6736(02)11911-8
8. Wu, Simiao et al. “Stroke in China: advances and challenges in epidemiology, prevention, and management.” The Lancet. Neurology vol. 18,4 (2019): 394–405. doi:10.1016/S1474-4422(18)30500-3
9. Mills, Katherine T et al. “Global Disparities of Hypertension Prevalence and Control: A Systematic Analysis of Population-Based Studies From 90 Countries.” Circulation vol. 134,6 (2016): 441–50. doi:10.1161/CIRCULATIONAHA.115.018912
10. Popkin, Barry M et al. “Cohort Profile: The China Health and Nutrition Survey–monitoring and understanding socio-economic and health change in China, 1989–2011.” International journal of epidemiology vol. 39,6 (2010): 1435–40. doi:10.1093/ije/dyp322
11. Yan, S et al. “The expanding burden of cardiometabolic risk in China: the China Health and Nutrition Survey.” Obesity reviews: an official journal of the International Association for the Study of Obesity vol. 13,9 (2012): 810–21. doi:10.1111/j.1467-789X.2012.01016.x
12. Cox, David R. "Regression models and life-tables." Journal of the Royal Statistical Society: Series B (Methodological) 34.2 (1972): 187–202.
13. Sesso, H D et al. “Systolic and diastolic blood pressure, pulse pressure, and mean arterial pressure as predictors of cardiovascular disease risk in Men.” Hypertension (Dallas, Tex.: 1979) vol. 36,5 (2000): 801-7. doi:10.1161/01.hyp.36.5.801
14. Benetos, Athanase et al. “Prognostic value of systolic and diastolic blood pressure in treated hypertensive men.” Archives of internal medicine vol. 162,5 (2002): 577–81. doi:10.1001/archinte.162.5.577
15. Zhao, Leilei et al. “Brachial pulse pressure and cardiovascular or all-cause mortality in the general population: a meta-analysis of prospective observational studies.” Journal of clinical hypertension (Greenwich, Conn.) vol. 16,9 (2014): 678–85. doi:10.1111/jch.12375

16. Mosley, William J 2nd et al. “Predictive utility of pulse pressure and other blood pressure measures for cardiovascular outcomes.” Hypertension (Dallas, Tex.: 1979) vol. 49,6 (2007): 1256-64. doi:10.1161/HYPERTENSIONAHA.106.083592
17. Rapsomaniki, Eleni et al. “Blood pressure and incidence of twelve cardiovascular diseases: lifetime risks, healthy life-years lost, and age-specific associations in 1·25 million people.” Lancet (London, England) vol. 383,9932 (2014): 1899 – 911. doi:10.1016/S0140-6736(14)60685-1
18. Du, Xin et al. “Association of Blood Pressure With Stroke Risk, Stratified by Age and Stroke Type, in a Low-Income Population in China: A 27-Year Prospective Cohort Study.” Frontiers in neurology vol. 10 564. 29 May. 2019, doi:10.3389/fneur.2019.00564
19. Cupples, L. A. "Some risk factors related to the annual incidence of cardiovascular disease and death using pooled repeated biennial measurements." Framingham Heart Study (1987).
20. Lew, E A. “High blood pressure, other risk factors and longevity: the insurance viewpoint.” The American journal of medicine vol. 55,3 (1973): 281–94. doi:10.1016/0002-9343(73)90130-7
21. Yano, K et al. “The impact of elevated blood pressure upon 10-year mortality among Japanese men in Hawaii: the Honolulu Heart Program.” Journal of chronic diseases vol. 36,8 (1983): 569–79. doi:10.1016/0021-9681(83)90145-5

Figures

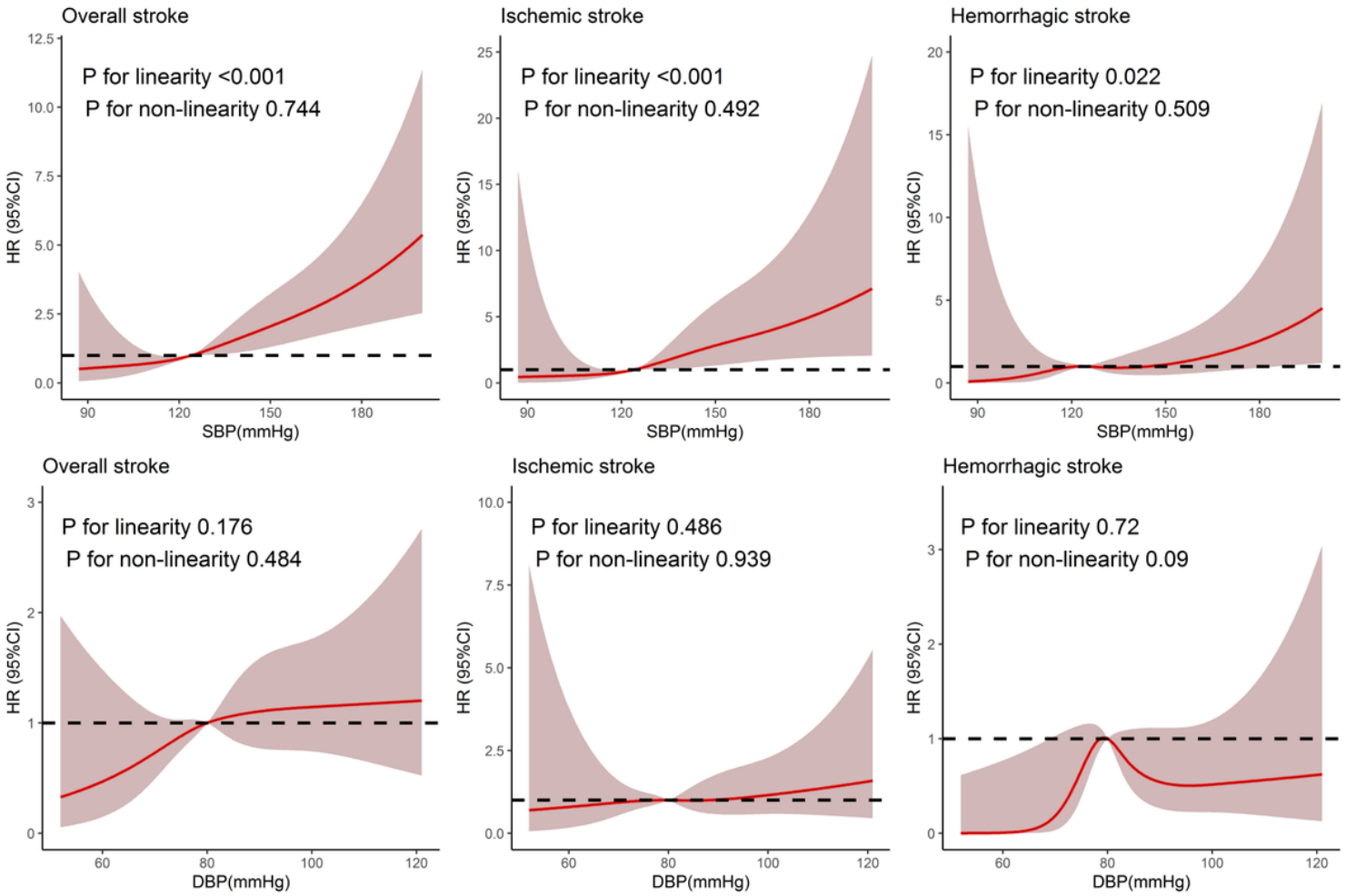

Figure 1

Association of BP with overall, ischemic and hemorrhagic stroke (model 3)

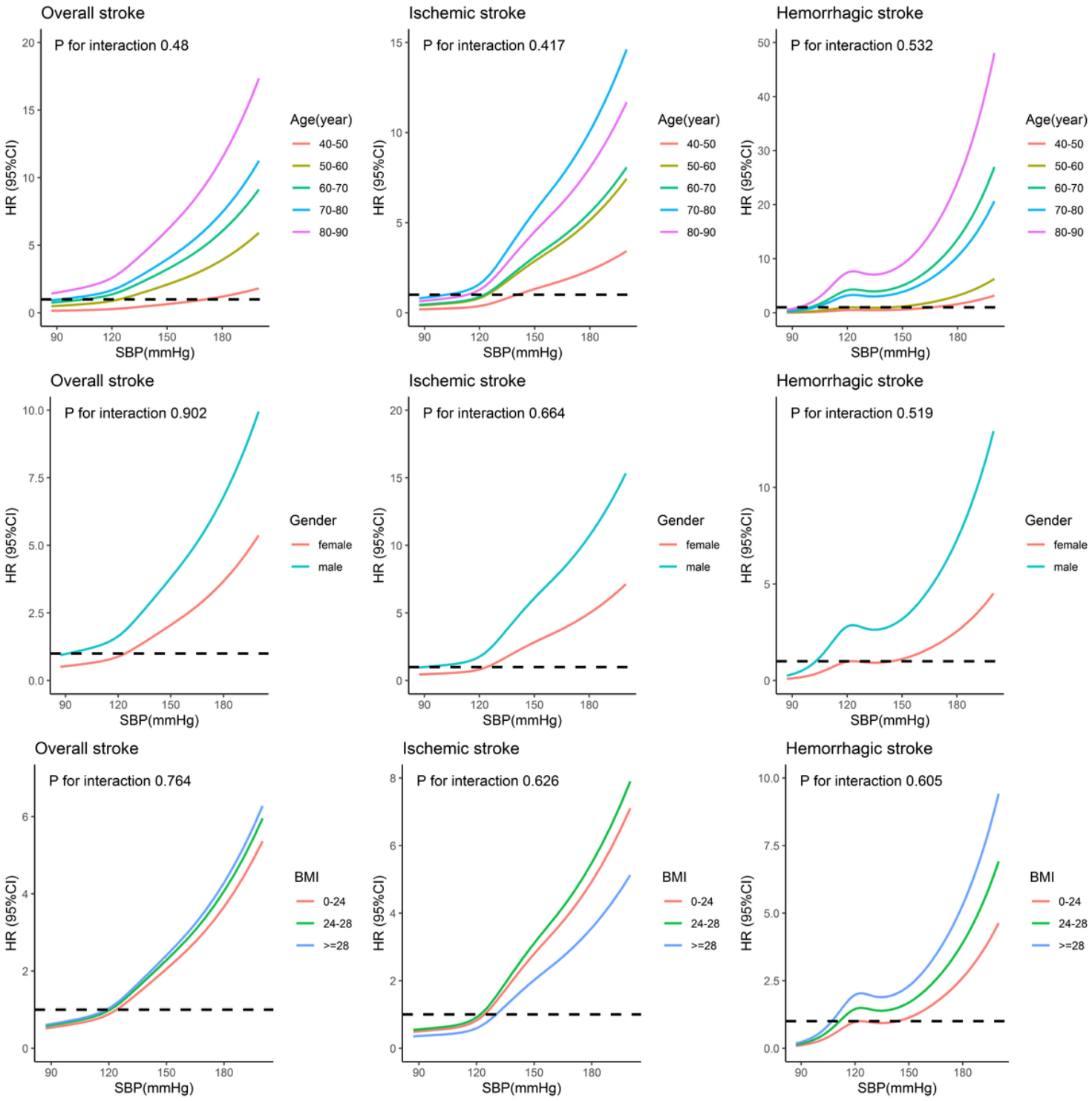

Figure 2

Association of BP with overall, ischemic and hemorrhagic stroke by age, gender and BMI (model 3)

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Appendix.docx