Title Page

Title: Effects of indoor and outdoor temperatures on blood pressure in a wintertime longitudinal study of Chinese adults.

Short title: Temperature and blood pressure in China

Authors: Talia J STERNBACH[a,b], Sam HARPER[a,b], Xiaoying LI[a], Xiang ZHANG[c], Ellison CARTER[d], Yuanxun ZHANG[e,f], Guofeng SHEN[g], Zhongjie FAN[h], Liancheng ZHAO[i], Shu TAO[g], Jill BAUMGARTNER[a,b]

Author affiliations:

[a] Department of Epidemiology, Biostatistics, and Occupational Health, McGill University, Montréal, QC, Canada

[b] Institute for Health and Social Policy, McGill University, Montréal, QC, Canada

[c] Department of Geography, McGill University, Montréal, QC, Canada

[d] Department of Civil and Environmental Engineering, Colorado State University, Fort Collins, CO, USA

[e] College of Resources and Environment, University of Chinese Academy of Sciences, Beijing, China

[f] CAS Center for Excellence in Regional Atmospheric Environment, Chinese Academy of Sciences, Xiamen, China

[g] College of Urban and Environmental Sciences, Laboratory for Earth Surface Processes, Sino-French Institute for Earth System Science, Peking University, Beijing, China

[h] Department of Medicine, Peking Union Medical College Hospital, Peking Union Medical College and Chinese Academy of Medical Sciences, Beijing, China

[i] National Center for Cardiovascular Disease, Fuwai Hospital, Peking Union Medical College and Chinese Academy of Medical Sciences, Beijing, China

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Corresponding authors and requests for reprints:

1. Sam Harper

   Address: 1020 Pine Avenue West, Montreal, QC 94305

   Email: sam.harper@mcgill.ca

   Phone: +1 514 398 2856

2. Shu Tao

   Address: Shaw Building No. 2, Peking University, Haidian District, Beijing, China

   Email: taos@pku.edu.cn

   Phone: +86 010-62751938

   Fax: 00 86 010-62751938

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Abstract

Objectives: We aimed to estimate the effects of indoor and outdoor temperature on wintertime blood pressure (BP) among peri-urban Beijing adults.

Methods: We enrolled 1,279 adults (ages: 40-89 y) and conducted measurements in two winter campaigns in 2018-19 and 2019-20. Study staff traveled to participant homes to administer a questionnaire and measure brachial and central BP. Indoor temperature was measured in the 5-min prior to BP measurement. Outdoor temperature was estimated from regional meteorological stations. We used multivariable mixed-effects regression models to estimate the within- and between-individual effects of indoor and outdoor temperature on BP.

Results: Indoor and outdoor temperatures ranged from 0.0 to 28.0˚C and −14.3 to 6.4˚C, respectively. In adjusted models, a 1˚C increase in indoor temperature was associated with decreased systolic BP [−0.4 mmHg, 95%CI: −0.7, −0.1 (between-individual; brachial and central BP); −0.5 mmHg, 95%CI: −0.8, −0.2 (within-individual, brachial BP); −0.4 mmHg, 95%CI: −0.7, −0.2 (within-individual, central BP)], diastolic BP [−0.2 mmHg, 95%CI: −0.4, −0.03 (between-individual); −0.3 mmHg, 95%CI: −0.5, −0.04 (within-individual)], and within-individual pulse pressure [−0.2 mmHg, 95%CI: −0.4, −0.04 (central); −0.3 mmHg, 95%CI: −0.4, −0.1 (brachial)]. Between-individual SBP estimates were larger among participants with hypertension. There was no evidence of an effect of outdoor temperature on BP.

Conclusions: Our results support previous findings of inverse associations between indoor temperature and BP but contrast with prior evidence of an inverse relationship with outdoor temperature. Wintertime home heating may be a population-wide intervention strategy for high BP and cardiovascular disease in China.

Key Words

Cardiovascular disease, Heating, Hypertension, Longitudinal studies, Temperature

Text

Introduction

Seasonal variability in outdoor temperature is associated with greater CVD mortality and non-fatal events, especially in winter [1, 2]. Changes in blood pressure (BP) may play an important role in these seasonal differences, though many factors may contribute [3]. Epidemiologic studies in Europe, East Asia, and North America showed greater temperature-related BP increases in winter and after colder outdoor temperatures, with larger effects among populations that are older age [4], spent less time indoors [5], and lived in places where central home heating is less common [4, 6].

The impacts of home heating and indoor temperature on cardiovascular risk are still poorly understood [7]. Studies in Europe found associations between excess winter deaths from CVD and poor housing conditions (i.e., lack of central heating or insulation) [8-10]. Seasonal differences in BP were smaller in Chinese adults living in regions with central heating compared with those without heating [4]. More recent studies in the U.K. and Japan reported inverse associations between indoor temperature and BP, and a trial of intensive room heating showed reductions in BP with home heating [11-13]. However, these studies and most others did not differentiate between indoor and outdoor temperature and are further limited by their cross-sectional designs [6].

The two studies [14, 15] of indoor and outdoor temperature on BP, both conducted in Japan, observed larger associations for indoor temperature. This distinction may be especially important in settings like China with a diversity of space heating practices, housing structures, and indoor thermal conditions, which can contribute to lower or unstable indoor temperatures

that may influence BP [9]. High BP poses a large and growing public health burden in China, where population levels of BP have steadily increased during the past several decades [16].

This study builds on the emerging hypothesis that exposures to low indoor and outdoor temperatures increase adult BP. Using data from a longitudinal study of 1,279 Chinese adults in peri-urban Beijing, China, we assessed the effects of indoor and outdoor temperatures on BP and investigated whether the outdoor temperature-BP associations were modified by indoor temperature.

Methods

1. Study location

The study was conducted in 50 villages located in 4 districts (Fangshan, Huairou, Mentougou, and Miyun) in the Beijing Municipality Region in northern China. Villages were selected for study because of a planned government-supported space heating program. Beijing has a temperate continental monsoon climate characterized by cold, dry winters and hot, humid summers. Access to central heating is limited in peri-urban Beijing where homes rely on coal or biomass-fueled heaters and *kangs* (a traditional Chinese combined cooking and heating stove) and electric-powered heaters [17].

2. Study design and data collection

Two data collection campaigns took place from December 2018 to March 2019 and from November 2019 to January 2020. In both campaigns, trained staff traveled to participants' homes to conduct tablet-based questionnaires, measure their BP, and place air pollution and temperature monitors in their homes. Anthropometrics (height, weight, and waist circumference) and whole blood samples were obtained several weeks later at a village clinic.

3. Study population

Participants were eligible for study if they were over 40 years old, maintained primary residence in the village, were not planning to move in the next year, and were not being treated with immunotherapy or corticosteroids. Simple random sampling of households was not logistically possible because many homes were vacant. Instead, village guides helped field staff to identify occupied households with eligible participants from a village roster. One participant

was selected at random from each home using a tablet-based randomization tool. We approached a census in some villages where a participant in every or nearly every occupied household was enrolled.

We recruited a total of 1,279 participants, including 997 and 1,111 participants in the first and second data collection campaigns, respectively (**Supplementary Fig. S1**). When a participant could not be reached (n = 110, 11%) or refused participation (n = 29, 3%) in the second campaign, another participant was randomly chosen from the remaining eligible individuals in the same household (typically the participant's spouse; n=80). Otherwise, we recruited a new participant from a different household in the same village (n=202). Participation rates in the first campaign ranged from 67-80% across the study villages. Participants provided written informed consent. Study protocols received ethical approval from Peking University (IRB00001052-18090) and McGill University (A08-E53-18B).

4. Blood pressure measurement and cardiovascular functions

Following 5-minutes of quiet rest, at least three brachial and central systolic (bSBP/cSBP) and diastolic (bDBP/cDBP) blood pressures (BPs) were taken by trained staff at 1-minute apart on the participant's supported right arm. We used an automated oscillometric device (BP+, Uscom Ltd, New Zealand) that estimates central pressures from the brachial cuff pressure fluctuations. The BP devices were calibrated by the manufacturer prior to the start of each campaign and previously validated against invasive cBP measurements [18, 19]. Up to five measurements were taken if the difference between the last two was >5 mmHg or staff were unable to obtain a reading. BP measurements were conducted in the participant's home and staff were trained on and followed strict quality control procedures, including use of an appropriately

sized cuff, correct positioning on the arm, and ensuring 5 minutes of quiet rest before measurement [20]. The average of the final two measurements was used for statistical analysis unless only one BP measurement was obtained (n=10 participants), in which case a single measurement was used. The time of day and day of the week were also recorded.

Peripheral (pPP) and central pulse pressures (cPP) were calculated by subtracting diastolic from systolic pressures, and pulse pressure amplification (PPamp) and SBP amplification (SBPamp) between the peripheral and central arteries were calculated as the ratio of pPP to cPP and bSBP to cSBP, respectively. Participants were considered hypertensive if systolic BP ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg or if they reported taking anti-hypertensive medication [21].

5. Indoor and outdoor temperature measurements

5.1. Indoor temperature

In all participant homes, indoor temperature (˚C) was measured during each household visit using a digital thermometer (Tianjianhuayi Inc., Beijing, China) in a centrally located room, away from heating sources and direct sunlight. Staff placed the probe in mid-air at a height that approximated the participant's shoulder height. Long-term indoor temperature was additionally measured for 6 to 12 months in a random subsample of 362 homes (37%) in the first campaign and 539 homes (50%) in the second campaign to evaluate whether indoor temperature in the minutes prior to BP assessment was representative of 'usual' daytime indoor temperature in the same month. In this subsample, staff placed temperature sensor (Thermochron® iButton® (DS1921G) or LabJack® Digit-THL®) on the interior wall of the room (height ~1.5m) where the participant reported spending the most time.

5.2. Outdoor temperature

We obtained hourly outdoor temperature data from 413 meteorological stations in Beijing and its neighboring provinces from the NOAA Integrated Surface Data database [22]. For each participant, we estimated their exposure to outdoor temperature on the day of BP measurement by inverse-distance weighting the hourly temperatures recorded by government meteorological stations within a 100 km radius of their home (typically 2-4 monitors). Estimated 24-h temperatures on the day of BP measurement were adjusted for altitude using the environmental lapse rate (equivalent to the dry adiabatic lapse rate) of $-6.5^{\circ}C$ per 1,000 meters.

6. Covariates

Staff administered structured questionnaires to collect information on household assets and energy use practices as well as participant demographics, health-related behaviors, previous diagnosis of health conditions including diabetes, current medication use, and self-reported history of cardiovascular events including heart disease, previous stroke, or a hospitalization in the past year. We used principal component analysis to construct a composite wealth index based on household assets.

Anthropometrics were obtained using a height ruler attached to a mechanical scale and SECA measuring tapes. Serum specimens were processed at a centralized testing facility for lipids (total cholesterol, high- and low-density lipoproteins, and triglycerides; mmol/L). In a random sub-sample of 489 participants (~50%) in the first campaign, we measured their personal exposures to fine particulate matter <2.5μm ($PM_{2.5}$) and black carbon, a pollutant indicator of

incomplete combustion, in the 24-hours after BP measurement. Details on air pollution exposure assessment are described elsewhere [23].

We based covariate selection on a directed acyclic graph that identified variables that could confound the models of temperature and blood pressure based on previous studies. Variables were included in statistical models as displayed in **Table 1**.

7. Statistical analysis

Mixed effects regression models with participant-specific random intercepts (ρ_{i0}) were used to estimate the effects of indoor and outdoor temperatures on blood pressure (**Equation 1**). Separate models were conducted for each exposure (indoor and outdoor temperature) and with each outcome (bSBP, cSBP, bDBP, cDBP, pPP, cPP, PPamp, SBPamp). In instances where one household occupant participated in the first campaign and a different household occupant participated in the second campaign (n=162 and 287 participants in campaigns 1 and 2, respectively), these participants were considered separately in the statistical models, each with their own intercept.

A cluster mean-centered term was used to estimate the average effect of a change in temperature on participants' BP between the first and second campaigns (i.e., the 'within-individual' effect; β_{1W}) [24]. Conditioning on an individual's mean value controls for observed and unobserved time-invariant individual characteristics (e.g., genetic factors, gender, some housing characteristics) that might confound the temperature-BP effect. Thus, we are leveraging variation in indoor temperature within the same households to estimate its effect on cardiovascular outcomes. The credibility of this effects rests on the assumptions that we have measured all time-varying confounders (i.e., covariates listed above) and there is no systematic

bias due to measurement error or selection bias [25]. A time-averaged, fixed effect term (β_{1B}) was used to estimate the between-individual difference in cardiovascular outcome per 1˚C change in temperature. Estimating both the within- and between-individual relationships can inform how temperature changes affect individuals' BP over time and how the temperature-BP relationship differs across a population, from one individual to next.

Natural cubic splines with 2 to 4 degrees of freedom (df) were used to investigate potential non-linear response functions between all continuous independent variables and BP. All temperature-BP relationships appeared linear. The relationship of age with DBP was modeled with df = 2. Fully adjusted regression models were fit with product terms for the interaction of indoor with outdoor temperature to allow for possible effect measure modification of the outdoor temperature-BP relationship by indoor temperature.

As sensitivity analyses, we fit models with an additional random intercept for the participant's village of residence to account for village-level clustering and investigated whether sex, hypertension status, or use of anti-hypertensive medication modified the temperature-BP relationships, based on findings from previous studies [4, 26]. We additionally adjusted for anti-hypertensive treatment, heart rate, exposure to air pollution, and blood lipid levels in our final multivariable models in the sub-sample of participants with these measurements (n=359 for air pollution; n=999 for blood lipids). These variables are risk factors for elevated BP [27, 28] that could differ by temperature but could also be mediating variables on the pathway between temperature and BP, where adjustment may lead to bias.

All analyses were conducted in RStudio [29]. The code to generate all tables and figures is available for download from the Open Science Foundation (https://osf.io/r28va/).

Results

1. Participant characteristics

This analysis included 1,256 participants (campaign 1 = 970; campaign 2 = 1,092; both campaigns = 806) with measured exposure to outdoor temperature and 1,255 participants (campaign 1 = 968; campaign 2 = 1,093; both campaigns = 806) with measured exposure to indoor temperature and each with at least one BP measurement. Multivariable models included 1,014 participants with complete covariate information (**Supplementary Fig. S2**).

Mean participant age was 60.3 y (SD = 9.0), 60% were female, and most (63%) were agricultural workers (**Table 1**). Participants in homes with warmer indoor temperatures were, on average, more likely to be married, female, never-smokers, and have more years of education. Mean BMI was 26.0 (SD = 3.7) kg/m^2 and did not differ for participants living in colder versus warmer homes. Average measures of BP, PP, and the proportion of participants with hypertension generally declined with increasing quintile of indoor temperature.

2. *Indoor and outdoor temperature*

Mean indoor temperature measured during household visits was 15.0˚C (range: 0 to 28˚C) and increased by 2.4˚C between the first and second campaigns. Mean outdoor temperature was identical across campaigns (−4.4˚C; range across campaigns: −14.3˚C to 6.4˚C). Indoor temperature at the time of BP measurement showed a Spearman correlation of 0.25 with outdoor temperature but was more highly correlated with long-term indoor measurements (Spearman r of 0.66, 0.59, and 0.53 with average indoor daytime temperature in the 24-h, 1 week, and 1 month after the household visit, respectively).

Most (79%) home temperatures were below the World Health Organization's recommended lower indoor temperature threshold (18˚C), which is based on the adverse health effects of cold outdoor temperatures [30]. The absolute difference between indoor and outdoor temperature for study homes varied by socio-demographic factors. Participants living in comparatively warmer homes were more likely to be in the highest wealth index quintile, report a greater number of total heating hours, primarily heat with electric-powered heaters, and have home insulation (**Supplementary Fig. S3**).

3. Effects of indoor and outdoor temperature on blood pressure

In multivariable models we found that a 1˚C increase in indoor temperature decreased brachial and central BP. The within-individual estimates for brachial and central SBP were −0.5 mmHg [95%CI: −0.8, −0.2] and −0.4 mmHg [95%CI: −0.7, −0.2], respectively, and the corresponding between estimates were both −0.4 [95%CI: −0.7, −0.1]. The within-individual estimates for brachial and central DBP were −0.3 mmHg [95%CI: −0.5, −0.04] and −0.2 mmHg [95%CI: −0.4, −0.01], respectively. Fully adjusted models generated an estimate for indoor temperature on cPP of −0.2 mmHg (95%CIs ranging from [−0.4, −0.04] to [−0.4, 0.01]) per 1˚C increase for the within- and between-individual effects, respectively. The effect of indoor temperature on within and between-individual pPP was −0.3 mmHg [95%CI: −0.4, −0.1] and −0.1 mmHg [95%CI: −0.3, 0.1] per 1˚C increase, respectively (**Figure 1A**).

The within- and between-individual effects of outdoor temperature on BP appeared linear and only showed small increases of 0.2 mmHg [95%CI: 0.01, 0.4] and 0.3 mmHg [95%CI: 0.1, 0.5] per 1˚C increase in outdoor temperature for central and peripheral between-individual PP,

respectively (**Figure 1B**). We did not observe consistent effects of outdoor temperature on other BP outcomes.

The inverse effects of indoor temperature on between-individual SBP were larger among participants with hypertension and on between-individual SBP and PP among participants taking anti-hypertensive medication (**Figure 2**; **Supplementary Table S6)**. Estimates were slightly attenuated for indoor temperature on SBP and larger for outdoor temperature after additionally controlling for exposure to air pollution (**Supplementary Table S7)**. Neither outdoor temperature nor sex modified the effect of indoor temperature on BP (**Supplementary Table S6**). No differences were observed after including a village-level random effect or additionally controlling for blood lipids, heart rate, or treatment with anti-hypertensive medication (**Supplementary Table S7**).

Discussion

In this longitudinal study of 1,256 peri-urban Beijing adults, exposure to higher indoor temperature had an inverse effect on brachial and central BP and pulse pressure, with larger BP effects among participants with hypertension and taking anti-hypertensive medication. We did not observe strong or consistent relationships between outdoor temperature and BP.

Our study has a number of important strengths that add to the existing literature on temperature and BP. We assessed the relative importance of both indoor and outdoor temperature for BP, which demonstrates the greater importance of indoor temperature and heating in our study setting, whereas prior studies investigated only one. Our panel design enabled us to investigate within-individual changes in exposure to temperature while controlling for measured and unmeasured time-invariant confounders, whereas most previous studies were cross-sectional. We also controlled for important time-varying confounders not included in most previous studies, including household wealth and participant demographics (i.e., marital status, education, occupation). Finally, the range of exposures to indoor temperatures in our study reflects those observed in regions of the world where central heating is unavailable [7] and is much wider than the indoor temperature ranges in Europe and North America where most studies of temperature and BP were conducted.

Our panel design strengthens our ability to estimate the effect of temperature on blood pressure in the absence of selection bias and measurement error. Simple random sampling of households was not possible because many households were unoccupied, but village guides were unaware of the research questions, limiting concern that selection into the study was correlated with temperature, BP, or covariates. Further, we approached a census of occupied homes in some villages and our median participant age and average BP levels are consistent with population-

based studies in peri-urban Beijing [31]. There was also little difference in BP or temperature between those who left the study after the first year, entered in the second year, and participated in both years (**Supplementary Table S8**). We implemented strict quality assurance and control procedures for study measurements, including rigorous staff training before each data collection campaign, and conducted routine maintenance checks on equipment to minimize the potential for measurement error.

The magnitudes of effect between indoor temperature and BP in our study fall within the range of reported associations in previous studies (SBP: −0.9 to −0.2 mmHg; DBP: −2.1 to −0.2 mmHg per 1˚C) [32, 33]. Our results are most similar to two recent studies of adults in the U.K. (systolic: −0.3 to −0.5 mmHg; diastolic: −0.2 to −0.5 mmHg per 1˚C) that, like our study, also controlled for socio-demographic confounders (e.g., education, wealth, marital status) [13, 34]. Controlling for these factors may be especially influential in settings like ours where socioeconomic constraints can influence home heating practices and exposure to outdoor temperature [7]. Our effects were slightly smaller than those observed in a study of rural Ghanaian adults exposed to a warmer indoor temperature range (30˚C to 45˚C) but did not control for socioeconomic status (−0.5 mmHg SBP and DBP per 1˚C increase) [35].

We did not observe consistent effects of outdoor temperature affects BP in our study, which contrasts with studies conducted mostly in Europe and North America with some evidence from East Asia and South Africa [32, 36]. In a population-based study in northern China, adults with central home heating had less seasonal variation in BP compared with adults with decentralized (i.e., room-specific) or no home heating [4]. Rural Japanese adults who spent an average of only 2 hours outdoors over a 48-hour period also showed an attenuated relationship of outdoor temperature with BP at temperatures below ~5-10˚C compared with the inverse

relationship observed at temperatures above 10˚C, which highlights the likely influence of indoor heating [37]. In comparison, the maximum outdoor temperature during BP measurement for our study was 6.4˚C and we observed greater daily variability in indoor compared with outdoor temperatures. It is possible that the largely null effects of outdoor temperature in our study are attributable to more time spent indoors in winter and household heating practices. Prior studies of the outdoor temperature-BP relationship that did not measure indoor temperature may have underestimated the true effect of temperature on BP and are also limited in drawing inference on the importance of indoor heating on BP management [15].

The hypothesized biological mechanism that drives cold-induced changes in BP is sympathetically-mediated vasoconstriction [38]. This reflex response is triggered with even modest reductions in environmental temperature that drive skin temperature below thermal neutral (~33-35˚C [39]), and results in reduced skin blood flow and increased arterial pressure [40]. BP can increase within minutes of exposure to colder temperatures and experimental studies show sustained BP increases for up to 6h after the initial exposure [41], particularly among older participants [38]. Evidence from animal studies suggests possible endothelial dysfunction from longer periods of cold exposure (4h per day over 8 weeks) that could lead to arterial damage and elevated BP [42]. The short-term mechanism is likely reflected in our study where indoor temperature was measured in the minutes before BP.

Consistent with previous studies in Chinese populations, we observed a stronger effect of indoor temperature on SBP among participants with hypertension and those taking anti-hypertensive medication [4, 26, 43]. Individuals with higher BP may have a pre-existing cardiovascular disease that impairs vasomotor function and may augment the vasoconstrictor response to colder temperatures [38]. Sex did not modify temperature-BP relationships in our

study whereas some previous studies found stronger effects in men and others found strong effects among women, possibly because of the interactive effects between sex and other baseline characteristics (e.g., smoking or alcohol consumption) on BP [44].

Moderate correlations between indoor and outdoor temperatures (spearman r = 0.20) suggest that indoor environments are influenced by participant heating behaviors and housing features. We observed warmer home temperatures of ~0.5 to 5.0˚C among participants who heated for more hours, used electric-powered heat pumps (compared with coal and/or biomass stoves), reported home insulation, and had greater wealth. However, these differences do not account for the combined effects of heating behaviors and housing conditions. If the effect of temperature on BP is causal, these variables indicate potential points of intervention to improve wintertime indoor temperatures and reduce population-level wintertime BP in rural and peri-urban China.

Our results correspond to an approximate 0.3% to 0.4% reduction in cSBP and bSBP, respectively, per 1˚C increase in indoor temperature, or a 3% to 4% decrease per 10˚C change. Various indoor heating programs and policies are currently under consideration across different regions of China, and our study indicates warmer, more stable indoor temperatures may act as an additional tool for BP regulation [45].

Our study has several limitations to consider in future studies. First, our stationary measures of indoor and outdoor temperature do not account for participant behavior and could result in exposure misclassification depending on participant location prior to BP measurements. Estimating the proportion of time that participants spend indoors versus outdoors may reduce this error but was logistically infeasible for our large study population [13]. Further, any measurement error should be non-differential and bias would likely be toward the null. The

short-term measurement of exposure may be an additional source of error in this study if BP is more strongly impacted by longer-term exposures to temperature. However, correlations of one-time and long-term temperature measurements indicate that the former are at least somewhat representative of the latter. Ambulatory BP monitoring may provide a better measurement to investigate the relationship with outdoor temperature but can be logistically challenging for larger studies like ours and more burdensome for participants.

Not all participants had complete covariate information, but those included in the multivariable analysis did not differ from the full sample in temperature exposure or BP. In such situations, we would not expect that potential bias would compromise an internally valid estimate. Finally, while our study included important time-varying covariates not captured in previous studies, we cannot rule out residual confounding from unmeasured time-varying variables in this observational study. We did not collect dietary data to assess possible changes in sodium intake across winter campaigns, though a recent study in peri-urban Beijing adults showed little difference in dietary intake of sodium over four years [46].

Conclusions

Our findings are consistent with an effect of indoor temperature on BP, but we do not find strong evidence of any effect of outdoor temperature. Our results further demonstrate the cardiovascular importance of maintaining warm indoor temperatures in winter in a setting with varied access to efficient space heating. These results are timely in consideration of current policies that target household heating and insulation in China, including ongoing programs to transition peri-urban homes to more efficient, electric-powered heaters. Such policies may provide a supplemental tool, in addition to lifestyle, dietary, and medication-based treatments, for population BP control and CVD prevention.

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Tables and Captions

$$BP_{it} = \alpha_{00} + \beta_{1W}(Temp_{it} - \overline{Temp_i}) + \beta_{1B}\overline{Temp_i} + \beta_2 z_{it} + \beta_3 z_i + (\rho_{i0} + \varepsilon_{it})$$

$$\rho_{i0} \sim N\left(0, \sigma^2_{Individual}\right)$$

Equation 1: Multivariable mixed effects regression model used to estimate the effects of temperature (Temp) on BP. Subscripts *i* and *t* indicate terms specific to each individual and study campaign (time), respectively; β_{1W} and β_{1B} give the mean-centered within-individual and time-averaged between-individual effects, respectively, for a 1˚C change in temperature on BP; $\beta_2 z_{it}$ represents the set of individual time-varying covariates; $\beta_3 z_i$ represents the set of individual time-invariant covariates; ρ_{i0} is the individual-specific random effect with the distribution ~ $N(0,\sigma_i^2)$;, and ε_{it} captures residual idiosyncratic error. α_{00} is the model intercept, representing the overall mean BP.

Table 1: Selected baseline participant characteristics by quintile of indoor temperature[a]. Reported as 'n (%)' for categorical variables and 'mean (SD)' for continuous variables. For categorical variables, the percent is the within-quintile percentage of participants.

		Indoor Temperature (*T*) Quintile (˚C)				
Participant characteristics	**Overall**	**1** $T \leq 11.3$	**2** $11.4 < T \leq 13.3$	**3** $13.4 < T \leq 15.2$	**4** $15.3 < T \leq 17.5$	**5** $T > 17.5$
n	1,255	253	263	238	250	251
Age, years	60.3 (9.0)	60.8 (8.6)	60.4 (9.0)	59.6 (9.5)	60.5 (9.0)	60.3 (9.0)
Sex (% female)	736 (58.9)	128 (51.2)	149 (57.1)	137 (57.6)	160 (64.0)	162 (64.5)
Marital status						
Married	1112 (89.0)	212 (84.8)	230 (88.1)	211 (88.7)	229 (91.6)	230 (91.6)
Divorced or separated	27 (2.2)	7 (2.8)	11 (4.2)	5 (2.1)	1 (0.4)	3 (1.2)
Widowed	99 (7.9)	25 (10.0)	18 (6.9)	21 (8.8)	18 (7.2)	17 (6.8)
Never Married	12 (1.0)	6 (2.4)	2 (0.8)	1 (0.4)	2 (0.8)	1 (0.4)
Education						
No school	141 (11.3)	37 (14.8)	27 (10.3)	28 (11.8)	26 (10.4)	23 (9.2)
Primary school	951 (76.1)	183 (73.2)	207 (79.3)	176 (73.9)	196 (78.4)	189 (75.3)
High school or greater	158 (12.6)	30 (12.0)	27 (10.3)	34 (14.3)	28 (11.2)	39 (15.5)
Current occupation						
Agriculture	786 (62.9)	165 (66.0)	169 (64.8)	140 (58.8)	160 (64.0)	152 (60.6)
Work outside the home	85 (6.8)	13 (5.2)	15 (5.7)	22 (9.2)	18 (7.2)	17 (6.8)
Work at home, retired, unemployed	311 (24.9)	54 (21.6)	66 (25.3)	64 (26.9)	62 (24.8)	65 (25.9)
Other/not stated	68 (5.4)	18 (7.2)	11 (4.2)	12 (5.0)	10 (4.0)	17 (6.8)
Asset-based wealth index[b]						
Lowest quintile (≤20%)	264 (22.1)	59 (24.0)	49 (19.2)	63 (27.4)	54 (22.9)	39 (17.1)
2nd quintile (20-40%)	214 (17.9)	43 (17.5)	53 (20.8)	44 (19.1)	35 (14.8)	39 (17.1)
3rd quintile (40-60%)	241 (20.2)	53 (21.5)	46 (18.0)	42 (18.3)	53 (22.5)	47 (20.6)
4th quintile (60-80%)	238 (19.9)	45 (18.3)	55 (21.6)	45 (19.6)	43 (18.2)	50 (21.9)
Highest quintile (>80%)	238 (19.9)	46 (18.7)	52 (20.4)	36 (15.7)	51 (21.6)	53 (23.2)
Environmental Tobacco Smoke						
Current smoker	334 (26.6)	81 (32.0)	72 (27.4)	68 (28.6)	62 (24.8)	51 (20.3)
Former smoker	161 (12.8)	29 (11.5)	44 (16.7)	30 (12.6)	26 (10.4)	32 (12.7)
Never smoker who lived with smoker	507 (40.4)	92 (36.4)	93 (35.4)	94 (39.5)	113 (45.2)	115 (45.8)
No exposure to tobacco smoking	253 (20.2)	51 (20.2)	54 (20.5)	46 (19.3)	49 (19.6)	53 (21.1)
Frequency of alcohol consumption						
Never	679 (54.1)	116 (45.8)	148 (56.3)	133 (55.9)	144 (57.6)	138 (55.0)
Occasional	251 (20.0)	51 (20.2)	50 (19.0)	50 (21.0)	47 (18.8)	53 (21.1)
Regular	86 (6.9)	19 (7.5)	16 (6.1)	17 (7.1)	14 (5.6)	20 (8.0)
Everyday	239 (19.0)	67 (26.5)	49 (18.6)	38 (16.0)	45 (18.0)	40 (15.9)
Exercise frequency						
Never	241 (19.2)	51 (20.2)	36 (13.7)	41 (17.2)	53 (21.2)	60 (23.9)
Occasional	79 (6.3)	16 (6.3)	16 (6.1)	17 (7.1)	15 (6.0)	15 (6.0)
Regular	244 (19.4)	51 (20.2)	56 (21.3)	40 (16.8)	50 (20.0)	47 (18.7)
Everyday	691 (55.1)	135 (53.4)	155 (58.9)	140 (58.8)	132 (52.8)	129 (51.4)
Outdoor temperature, ˚C	-4.5 (4.0)	-6.8 (3.7)	-4.9 (4.3)	-4.1 (3.9)	-3.1 (3.8)	-3.5 (3.2)
Personal exposure[d]	132.5 (245.3)	163.3 (268.3)	106.4 (126.2)	111.4 (144.7)	160.7 (427.1)	99.8 (99.8)

$PM_{2.5}$, $\mu g/m^3$						
Black carbon, $\mu g/m^3$	4.0 (5.4)	5.4 (6.5)	3.5 (5.4)	3.3 (3.9)	3.9 (5.6)	2.9 (2.8)
BMI, kg/m²	26.0 (3.7)	25.8 (3.8)	26.2 (3.5)	26.2 (3.8)	25.8 (3.4)	26.0 (3.8)
Waist circumference, cm	87.3 (10.1)	87.0 (10.2)	88.0 (10.4)	87.3 (9.9)	86.4 (9.4)	87.7 (10.6)
Total cholesterol, mmol/L	4.8 (0.9)	4.8 (1.0)	4.7 (0.9)	4.8 (1.0)	4.8 (0.9)	4.7 (0.9)
Triglycerides, mmol/L	1.5 (1.0)	1.5 (1.0)	1.6 (1.1)	1.5 (0.8)	1.5 (1.1)	1.7 (1.0)
High-density lipoprotein cholesterol, mmol/L	1.4 (0.4)	1.4 (0.4)	1.4 (0.3)	1.4 (0.4)	1.4 (0.3)	1.4 (0.4)
Low-density lipoprotein cholesterol, mmol/L	3.0 (0.9)	3.0 (0.9)	3.0 (0.9)	3.1 (0.9)	3.1 (0.9)	2.9 (0.8)
Heart rate, bpm	72.4 (10.8)	72.3 (10.7)	71.6 (10.2)	72.1 (10.5)	73.0 (10.8)	72.9 (11.7)
Central systolic BP, mmHg	123.9 (15.6)	126.0 (16.7)	126.6 (15.3)	122.39 (15.08)	122.56 (15.89)	121.9 (14.6)
Central diastolic BP, mmHg	83.0 (11.7)	84.6 (12.7)	84.2 (10.6)	81.98 (11.38)	81.75 (11.79)	82.2 (11.6)
Brachial systolic BP, mmHg	129.7 (15.8)	131.8 (16.6)	131.9 (15.6)	128.2 (15.6)	128.8 (15.9)	127.6 (15.0)
Brachial diastolic BP, mmHg	82.0 (11.5)	83.8 (12.4)	83.3 (10.5)	80.8 (11.3)	80.8 (11.4)	81.2 (11.5)
Central PP, mmHg	41.0 (10.5)	41.4 (10.4)	42.7 (10.8)	40.4 (10.1)	40.8 (11.1)	39.6 (9.6)
Peripheral PP, mmHg	47.7 (10.9)	48.0 (10.5)	48.7 (10.8)	47.4 (10.8)	48.0 (11.9)	46.4 (10.5)
PP amplification, mmHg	1.2 (0.1)	1.2 (0.1)	1.2 (0.1)	1.2 (0.1)	1.2 (0.1)	1.2 (0.1)
SBP amplification, mmHg	1.1 (0.1)	1.1 (0.1)	1.0 (0.03)	1.1 (0.03)	1.1 (0.1)	1.1 (0.03)
Hypertension[c]	737 (58.7)	153 (60.5)	165 (62.7)	134 (56.3)	143 (57.2)	142 (56.6)
Anti-hypertensive medication use	525 (82.4)	99 (79.2)	109 (80.1)	97 (79.5)	118 (88.7)	102 (84.3)
Area of home that is heated, m²	81.3 (49.2)	77.1 (48.1)	80.4 (45.4)	84.6 (55.4)	78.9 (46.9)	85.7 (50.0)
Wall insulation						
No walls insulated	263 (25.3)	50 (25.1)	63 (28.8)	41 (20.3)	56 (28.3)	53 (23.8)
Some walls insulated	523 (50.2)	107 (53.8)	106 (48.4)	107 (53.0)	90 (45.5)	113 (50.7)
All walls insulated	255 (24.5)	42 (21.1)	50 (22.8)	54 (26.7)	52 (26.3)	57 (25.6)
Window insulation						
Wood-framed windows	11 (1.1)	1 (0.5)	4 (1.9)	2 (1.0)	4 (2.1)	0 (0.0)
Single-pane windows	347 (34.3)	67 (34.0)	76 (35.8)	71 (36.8)	61 (31.8)	72 (32.9)
Double-pane windows	655 (64.7)	129 (65.5)	132 (62.3)	120 (62.2)	127 (66.1)	147 (67.1)
Roof insulation (% with insulation)	144 (13.8)	20 (10.1)	30 (13.7)	28 (13.9)	24 (12.1)	42 (18.8)
Daily household heating hours, room*hours/day	76.5 (62.3)	65.1 (65.3)	75.2 (57.9)	80.5 (62.8)	76.1 (61.4)	85.8 (62.5)

BMI=body mass index; BP=blood pressure; PP=pulse pressure; T=temperature

[a] 'Baseline' refers to the first measurement for the participant.

[b] The wealth index is presented here in quintiles but included as a continuous variable in regression models.

[c] Hypertension defined as currently taking anti-hypertensive medication, and/or SBP >= 140, and/or DBP >= 90 mmHg [21].

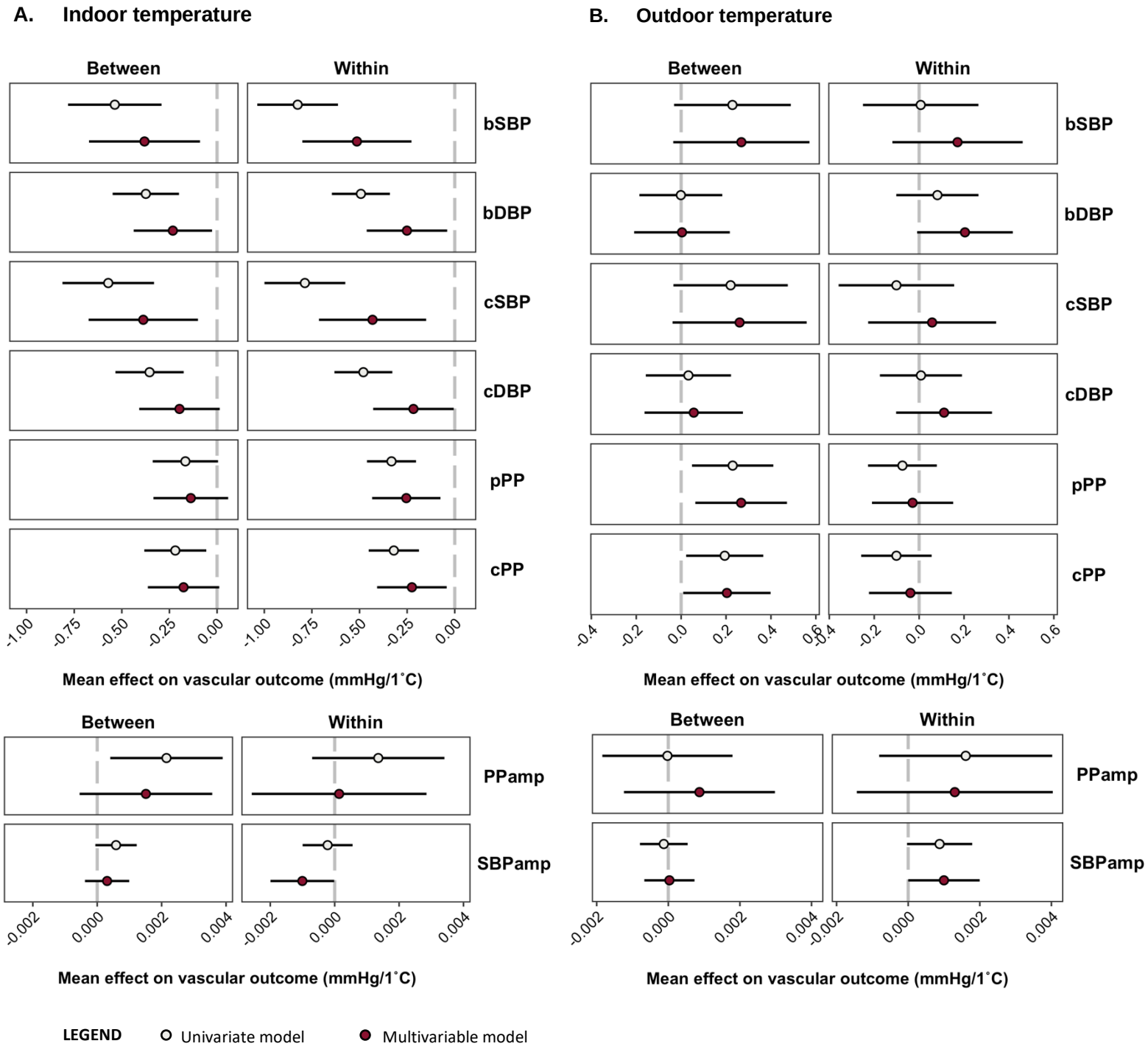

Figure 1: Changes in brachial and central blood pressure and pulse pressure per 1˚C increase in (A) indoor temperature and (B) outdoor temperature in Chinese adults.

The average effect for each outcome and their 95% confidence intervals is shown for the univariate and multivariable models. Effect sizes and 95% confidence intervals for each model are reported in **Supplementary Table S4**. Note different x-axis scales for PPamp and SBPamp.

Multivariable models are adjusted for age, sex, hour of BP measurement (categorical), day of BP measurement, BMI, waist circumference, diabetes diagnosis, frequency of alcohol consumption, exposure to environmental tobacco smoke, exercise frequency, farming frequency, occupation, education, household wealth index, marital status, district of residence, and outdoor temperature (indoor temperature models only).

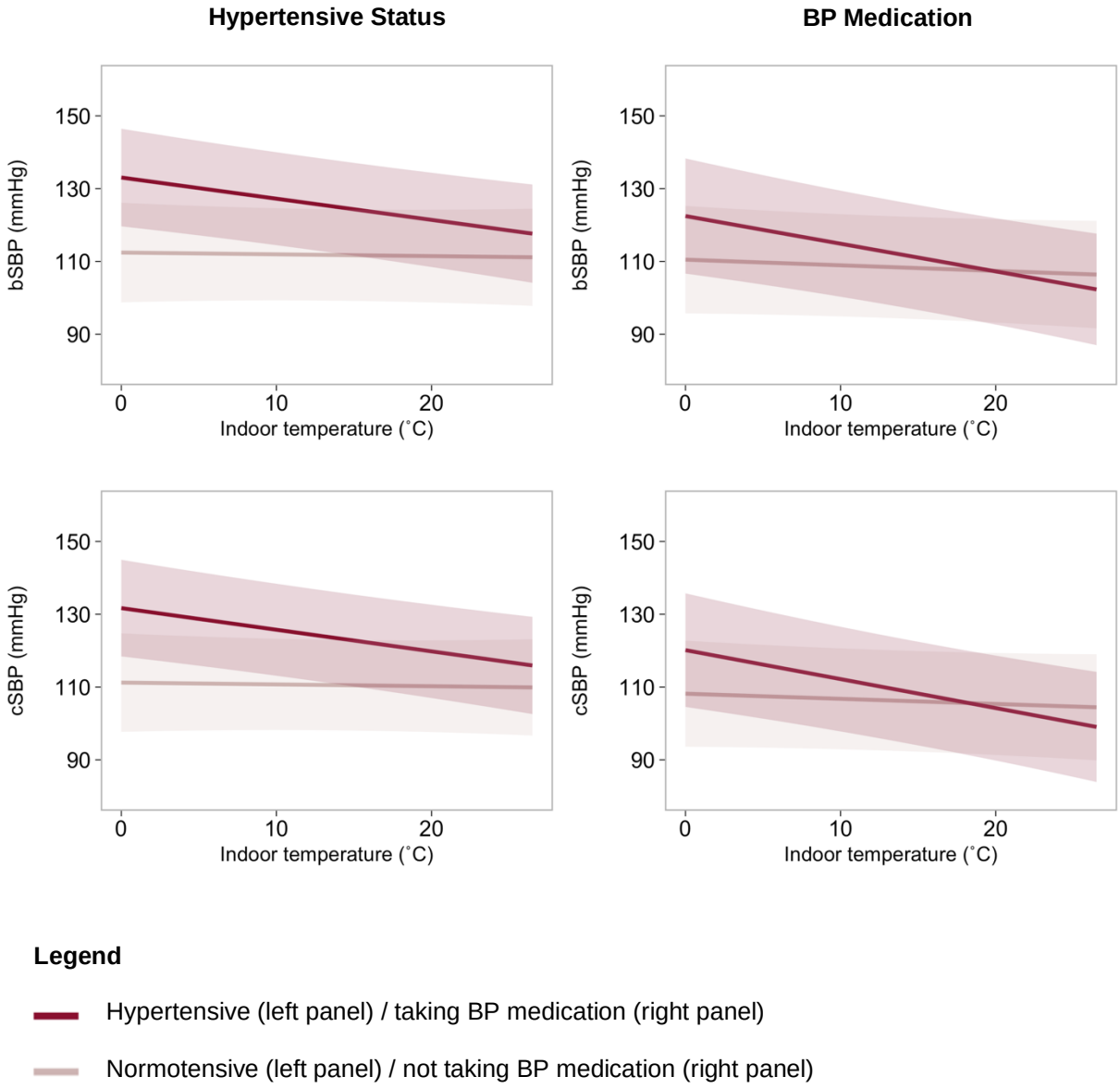

Figure 2: Effects of between-individual changes in indoor temperature on brachial (bSBP) and central (cSBP) systolic BP by hypertensive status (left) and treatment with BP medication (right). The dark pink line shows the effect for hypertensive participants (left panel) or participants taking anti-hypertensive medication (right panel) while the light pink line shows the effect for normotensive participants (left panel) or participants not

taking anti-hypertensive medication (right panel). The product terms for these measurements and 95% confidence intervals are reported in **Supplementary Table S6**.

Supplemental Digital Content

Supplemental Digital Content File 1: SI_TempBP_Submission.docx