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**FULL RESEARCH PAPER**
*Arterial hypertension*

# Variations in blood pressure after a 75 g oral glucose load and their implications for detecting hypertension and postprandial hypotension in Chinese adults: a cross-sectional study

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**Aims** Current hypertension guidelines fail to discriminate between fasting and postprandial blood pressure (BP) measurements. Meal ingestion often triggers a marked increase in splanchnic blood flow, potentially inducing a sustained fall in systolic BP of ≥20 mmHg, termed postprandial hypotension (PPH). This study aimed to evaluate BP responses to a 75 g glucose drink and its implications for detecting hypertension and PPH in community-dwelling adults.

**Methods and results** A stratified multi-stage random sampling method was used to obtain a nationally representative sample of $n = 4429$ adult residents between April 2020 and January 2021 in China. BP and heart rate (HR) were measured before, and 1 and 2 h after, a 75 g glucose drink. When fasting, 38.4% of the study population had high BP (BP ≥140/90 mmHg). Following the glucose drink, SBP and DBP decreased (SBP by 6.2 [95% CI: 5.8, 6.6] mmHg and 8.1 [7.7, 8.5] mmHg, DBP by 4.7 [4.4, 4.9] mmHg and 6.1 [5.8, 6.4] mmHg), and HR increased (by 4.3 [4.0, 4.5] bpm and 2.6 [2.4, 2.9] bpm) at 1 and 2 h ($P < 0.001$ for all), with only 30.9% and 27.0% of the study population having high BP at 1 and 2 h, respectively. After adjustment for age and sex distribution, 19.9% of the general population was estimated to have PPH. Postprandial hypotension was associated with an increased risk of combined cardiovascular disease and stroke.

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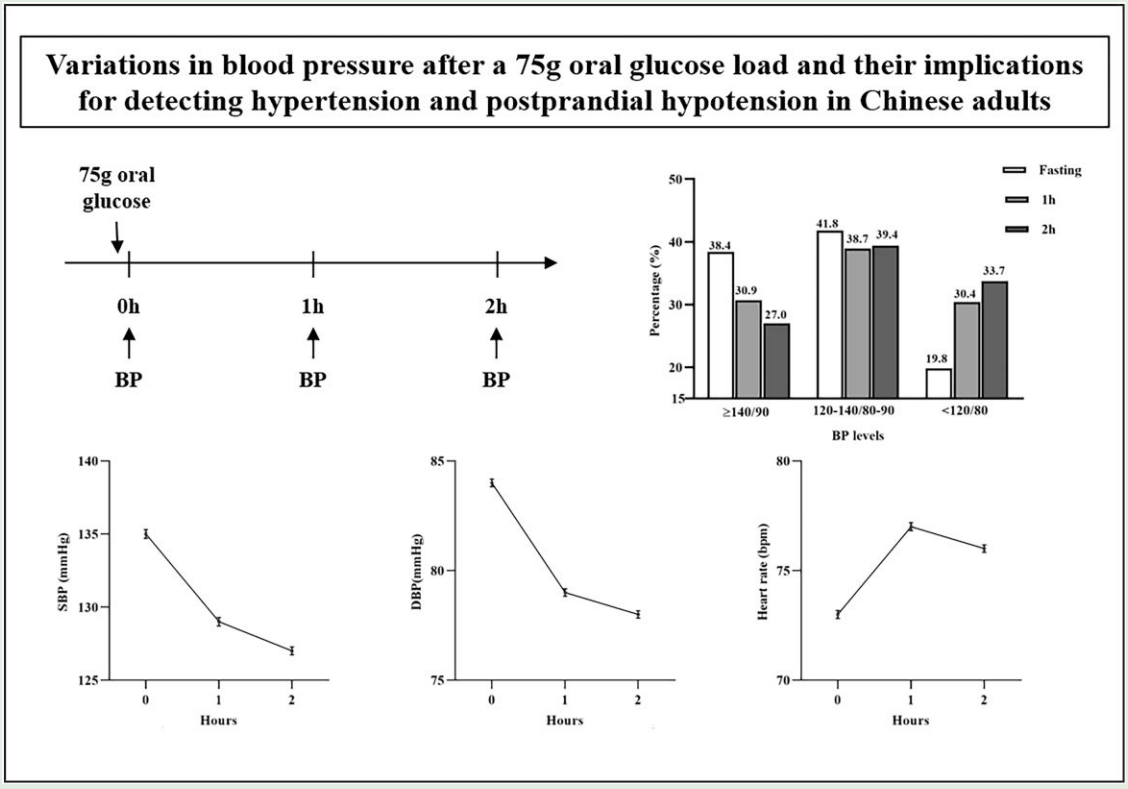
**Conclusion** Ingestion of a 75 g glucose drink often lowers BP, frequently leading to PPH and influencing the detection of hypertension. Accordingly, guidelines for measurements of BP and interpretation of outcomes should consider the potential impact of meal ingestion on BP.

**Lay summary**

- Meal ingestion increases blood pooling into the gut, which may result in a sustained fall in systolic blood pressure (BP) and, in some cases, with a magnitude ≥20 mmHg, known as postprandial hypotension (PPH).
- This cross-sectional study showed in community-dwelling Chinese adults that, after a 75 g glucose drink, both systolic BP and diastolic BP decreased, and heart rate increased, with a reduced detection rate of hypertension and an estimated rate of PPH of 19.9%.
- Guidelines for measurements of BP and interpretation of outcomes should consider the potential impact of meal ingestion on BP.

**Graphical Abstract**




**Keywords** Blood pressure • Hypertension • Postprandial hypotension • Oral glucose tolerance test

# Introduction

Normalization of elevated blood pressure (BP) is pivotal in minimizing the risk of cardiovascular events, including stroke, and reducing mortality. For example, death from stroke, heart disease, and other vascular disorders approximately doubles with every 20 mmHg increase above 115 mmHg in systolic blood pressure (SBP) and every 10 mmHg increase above 85 mmHg in diastolic blood pressure (DBP),[1] while stringent control of BP, including the attainment of SBP within the range of 110–130 mmHg[2] or the maintenance of SBP within the target range of 120–140 mmHg over long term,[3] has been reported to substantially reduce cardiovascular events.

Precise measurement of BP represents the initial step in the management of hypertension. Current guidelines for the diagnosis and management of hypertension provide recommendations relating to measurement procedures, and the potential impact of different settings on BP measurements (e.g. office vs. home), and emphasize the need to evaluate BP at specific times throughout the day and night, particularly with the use of ambulatory equipment.[4–10] However, while some guidelines suggest that home BP monitoring should be undertaken following an overnight fast, none provide specific advice about measurement of BP in relation to meals,[4–10] which potentially represents a major omission.

Meal ingestion triggers a series of haemodynamic responses, characterized by a marked increase in splanchnic blood flow, consequent

reduction in peripheral blood volume, and the necessity for compensatory increases in cardiac output (reflected by increases in heart rate [HR]) and systemic vascular resistance to prevent a fall in BP.[11] If these compensatory mechanisms are impaired, postprandial hypotension (PPH), defined as a fall in SBP of ≥20 mmHg within 2 h after the start of a meal or a glucose drink, or to <90 mmHg when baseline SBP is over 100 mmHg, may occur.[12] Hitherto, there is no consensus on the choice of a 'test meal' for the detection of PPH, but in many cases, the diagnostic 'meal' for PPH has been a glucose drink, as used in the oral glucose tolerance test for the diagnosis of type 2 diabetes and impaired glucose tolerance.[11] PPH has been shown to occur much more frequently than previously appreciated, with a high prevalence being reported in studies involving 'healthy' older people (~20%), hospitalized older patients (20–91%), nursing home residents (24–38%), individuals with hypertension (27.4–72.8%), and in long-standing type 1 and type 2 diabetes (~40%).[11] PPH also appears to occur more frequently than orthostatic hypotension, which is more widely recognized.[13] Importantly, PPH strongly predisposes to syncope, falls, coronary events, stroke, and mortality.[14,15] Evidence derived from cohort studies indicates that PPH is associated with an increased risk of cardiovascular disease (CVD) events as well as mortality in the elderly.[16,17] Moreover, a greater reduction in postprandial SBP is associated with a higher rate of both incident coronary events and total mortality.[15] A recent meta-analysis indicates that PPH is associated with a 52% increase in the risk of combined CVD outcomes and all-cause mortality.[18] These observations attest to the need to evaluate both fasting and postprandial BP in the diagnosis and management of hypertension.

To our knowledge, the BP response to a nutrient load and its determinants have not been evaluated in the general population. Moreover, the prevalence of PPH, its risk factors, and association with CVD have only been evaluated in specific populations with relatively small sample sizes.[11,18] The aim of this cross-sectional study was, accordingly, to determine the effects of a standardized nutrient load (75 g oral glucose) on BP and HR, as well as the prevalence and determinants of PPH, in a community-dwelling adult population.

# Methods

## Study population

A cross-sectional study was conducted between April 2020 and January 2021 in China, which was embedded in the second follow-up of the SENSIBLE-cohort study (Study on Evaluation of iNnovated Screening tools and determInation of optimal diagnostic cut-off points for type 2 diaBetes in Chinese muLti-Ethnic), designed to determine the optimal cut-off values for advanced glycation end-products and glycated haemoglobin (HbA1c) for diagnosing type 2 diabetes in China.[19] To select a nationally representative sample of adults in the general population, a multi-stage cluster and simple randomization method was used. In Stage 1, the sampling process was stratified according to geographic region (North, South, East, and West China), and six provinces, including Jilin (North), Yunnan and Guangxi (South), Fujian and Jiangsu (East), and Xinjiang Uyghur autonomous region (West), were selected. In Stage 2, cities in each province were numbered and selected (1–2 cities in each province) using a simple random sampling method, yielding a total of eight cities. In each city, 1 neighbourhood community and 1 administrative village were selected by a simple random sampling method. Accordingly, the study involved participants of six different ethnicities, including Han, Zhuang, Uygur, Kazak, Dai, and Korean. Finally, individuals who were ≥18 years old and had lived in their current residence for ≥5 years were eligible to participate and were stratified according to sex and age. Individuals who were pregnant, or had a history of diabetes, significant mental illness or a condition which precluded them from completing the study procedures, were excluded.

A total of 5000 subjects were invited, of whom 4511 participated, with an overall response rate of 90.2% (81.8% [1607/1964] for males and 95.7% [2904/3036] for females). After excluding 40 participants with missing data on age or sex and 42 participants with incomplete BP measurements due to technical problems, a total of 4429 participants were included in the final analysis (see Supplementary material online, *Figure S1*). The research protocol was approved by the Human Research Ethics Committee of Zhongda Hospital, Southeast University, Nanjing, China. All participants provided written informed consent.

## Data collection

Participants were instructed to maintain their usual lifestyle for at least 3 days, and to fast overnight for a minimum of 10 h prior to being studied. Smoking was prohibited from the night before the study. Anti-hypertensive medications scheduled for the night before or morning of the study were withheld. On the study day, baseline BP and HR were measured on the non-dominant arm using a validated automated sphygmomanometer (YE900, Yuwell, China)[20] after participants had been seated in a quiet environment for at least 10 min (feet on floor, back supported). Vigorous exercise was avoided for at least 24 h before the study visit. The cuff size was chosen according to the arm circumference of each participant and was positioned on the upper non-dominant arm at the level of the heart. A fasting venous blood sample was then obtained for measurements of HbA1c and fasting plasma glucose (FPG), triglycerides (TG), total cholesterol (TC), high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, alanine aminotransferase (ALT), aspartate aminotransferase (AST), γ-glutamyl transpeptidase (GGT), total bilirubin (Tbil), uric acid (UA), creatinine (Cr), and blood urea nitrogen (BUN). A fasting spot urine sample was collected for measurements of albumin and creatinine. A standardized questionnaire was used to obtain information relating to sociodemographic characteristics and medical history. Body weight, height, waist circumference, and hip circumference were also measured. Body mass index (BMI) was calculated as body weight (kg) divided by height squared (m), and waist-hip ratio (WHR) was calculated as waist circumference (cm) divided by hip circumference (cm). Participants then ingested a 200 mL drink containing 75 g glucose. Measurements of BP and HR were repeated at 1 and 2 h after the drink. Due to logistical constraints, a single measurement was conducted in a consistent manner at each timepoint. A second venous blood sample was obtained for measurement of the 2h plasma glucose concentration (2h-PG). All the data were collected via standardized procedures by well-trained investigators.

All blood samples were processed on site within 30 min of collection, and conveyed by air in iceboxes to a central laboratory (Nanjing Adicon Clinical Laboratories) and analysed immediately upon arrival using high-performance liquid chromatography (D-10™Hemoglobin Analyzer, Bio-Rad Inc., CA, USA) for HbA1c, and an automated chemistry analyzer (SynchronLX-20, Beckman Coulter Inc., CA, USA) for all other measurements.

## Definitions

As previously, PPH was defined as a fall in SBP ≥20 mmHg within 2 h after the glucose drink.[12] Both males and females were classified by BMI as lean (<24 kg/m$^2$), overweight 24–27.9 kg/m$^2$, or obese ≥28 kg/m$^2$.[21] Hypertension was defined as a baseline SBP ≥140 mmHg, diastolic DBP ≥90 mmHg, current use of anti-hypertensive medication, or previously diagnosed hypertension.[8] Glycaemic status was classified according to the 2020 Chinese Diabetes Society criteria:[22] diabetes was defined as a FPG ≥7.0 mmol/L, 2h-PG ≥11.1 mmol/L, HbA1c ≥ 6.5%, or a self-reported history of diabetes; prediabetes as impaired fasting glucose (FPG between 6.1 and 6.9 mmol/L) or impaired glucose tolerance (2h-PG between 7.8 and 11.0 mmol/L); and normoglycaemia as FPG <6.1 mmol/L and 2h-PG <7.8 mmol/L. The presence of macrovascular disease was determined by a self-reported history of CVD or stroke,[23] and micro-vascular disease by the presence of retinopathy (diagnosed according to the Early Treatment Diabetic Retinopathy Study criteria)[24] or microalbuminuria (diagnosed by a urine albumin: creatinine ratio > 30 mg/g).[22] Dyslipidaemia was defined as

**Table 1 Demographic and biochemical parameters in the study population**[a]

| Characteristic | Overall ($n = 4429$) | Male ($n = 1547$) | Female ($n = 2882$) |
|---|---|---|---|
| Age (year) | 56.8 ± 9.8 | 57.8 ± 10.0 | 56.3 ± 9.6 |
| BMI (kg/m$^2$) | 25.25 ± 3.83 | 25.26 ± 3.77 | 25.24 ± 3.86 |
| Fasting SBP (mm Hg) | 135 ± 20 | 135 ± 20 | 135 ± 20 |
| Fasting DBP (mm Hg) | 84 ± 12 | 85 ± 12 | 83 ± 12 |
| Fasting HR (bpm) | 73 ± 12 | 72 ± 12 | 73 ± 12 |
| FPG (mmol/L) | 5.35 ± 0.78 | 5.40 ± 0.78 | 5.32 ± 0.79 |
| 2h-PG (mmol/L) | 7.35 ± 2.36 | 7.34 ± 2.40 | 7.35 ± 2.34 |
| HbA1c (%) | 5.63 ± 0.67 | 5.64 ± 0.76 | 5.62 ± 0.61 |
| TC (mmol/L) | 4.69 ± 0.95 | 4.61 ± 0.92 | 4.74 ± 0.96 |
| TG (mmol/L) | 1.26 (0.92, 1.84) | 1.24 (0.90, 1.81) | 1.23 (0.87, 1.74) |
| HDL-c (mmol/L) | 1.42 ± 0.28 | 1.39 ± 0.29 | 1.44 ± 0.28 |
| LDL-c (mmol/L) | 2.62 ± 0.70 | 2.58 ± 0.68 | 2.65 ± 0.70 |
| UA (mmol/L) | 297 ± 84 | 313 ± 84 | 288 ± 82 |
| Cr (umol/L) | 57 ± 14 | 60 ± 14 | 56 ± 13 |
| eGFR (mL/min/1.73 m$^2$) | 103.6 ± 12.6 | 103.5 ± 12.3 | 103.6 ± 12.8 |
| BUN (umol/L) | 5.1 ± 1.4 | 5.2 ± 1.4 | 5.0 ± 1.4 |
| ALT (U/L) | 15 (10, 21) | 15 (11, 23) | 14 (10, 21) |
| AST (U/L) | 21 (18, 26) | 22 (18, 26) | 21 (18, 26) |
| GGT (U/L) | 19 (13, 29) | 20 (14, 34) | 18 (13, 27) |
| Tbil (umol/L) | 10.0 (7.7, 13.2) | 10.6 (8.1, 14.4) | 9.7 (7.5, 12.7) |
| Hypertension ($n$ [%]) | 1982 (44.8) | 713 (46.1) | 1269 (44.0) |
| Diabetes ($n$ [%]) | 545 (12.3) | 211 (13.6) | 334 (11.6) |
| Dyslipidaemia ($n$ [%]) | 950 (21.4) | 355 (22.9) | 595 (20.6) |
| CKD ($n$ [%]) | 26 (0.6) | 8 (0.5) | 18 (0.6) |
| Hepatic dysfunction ($n$ [%]) | 109 (2.5) | 51 (3.3) | 58 (2.0) |

[a]Some values were missing, including $n = 18$ for body mass index (BMI), $n = 17$ for fasting heart rate (HR), $n = 64$ for 2 h plasma glucose (2h-PG), $n = 9$ for glycated haemoglobin (HbA1c), $n = 2$ for fasting plasma glucose (FPG), total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), uric acid (UA), estimated glomerular filtration rate (eGFR) and blood urea nitrogen (BUN), $n = 4$ for alanine aminotransferase (ALT), aspartate aminotransferase (AST), γ-glutamyl transpeptidase (GGT), and total bilirubin (Tbil).

either high TC (≥6.20 mmol/L), high TG (≥2.30 mmol/L), low HDL cholesterol (<1.00 mmol/L), or high LDL cholesterol (≥4.10 mmol/L); chronic kidney disease was defined as a reduced eGFR (<60 mL/min/1.73m$^2$), calculated by the CKD-EPI formula;[25] hepatic dysfunction was defined as an elevation of liver enzymes at least twice the upper limit of the normal reference range (ALT ≥80 U/L, AST ≥100 U/L, GGT ≥120 U/L, Tbil ≥40 μmol/L).[26]

## Statistical analysis

The sample size was estimated on the basis of the cross-sectional design of the study. If the estimated prevalence of PPH in the general population is approximately 20%, with the maximum allowable error at 2% and the design effect at 2, a minimum of $n = 3073$ participants would be required. To allow for a response rate of 90%, the minimum sample size would be 3414. Categorical data are presented as percentages, and continuous data as mean ± standard deviation or median (25th–75th percentile). Repeated-measures ANOVA was used to compare BP and HR responses to oral glucose. Factors associated with changes in BP and HR following the glucose load were analyzed using mixed linear models, with time as the fixed effect, subject as random effect, and age, sex, and ethnicity as co-variates. Subjects were further stratified according to age (below and above the median age of 58 years), sex, and microalbuminuria status to evaluate factors associated with changes in SBP, DBP, and HR. The overall prevalence of PPH was adjusted for age and sex according to the 2020 Chinese population census.[27] Estimates of the prevalence of PPH in age groups <40 years, 40–49 years, 50–59 years, 60–69 years, and ≥70 years were weighted according to sex distribution, and estimates stratified by sex were adjusted according to the population proportions in age groups <40 years, 40–49 years, 50–59 years, 60–69 years, and ≥70 years. Crude rates of PPH were first stratified by BMI (lean, overweight, or obese), hypertension (present or not), and glycaemic status (normoglycaemia, prediabetes, or diabetes). Because PPH is frequently a reflection of an inadequate compensatory increase in HR, we also examined the associations between the changes in SBP and HR at both 1 and 2 h after oral glucose in participants with and without PPH using Pearson correlation analysis. Logistic regression analyses were then used to assess the association of PPH with macrovascular and micro-vascular disease, with adjustment for age, sex, ethnicity, BMI, hypertension, HbA1c, TC, TG, and eGFR. Because the number of missing values for co-variates considered in the regression analysis was modest (<10%), substitution of missing values was not conducted in the analyses. A two-sided $P$-value of <0.05 was considered significant. All statistical analyses were conducted using SPSS 25.0 (Chicago, IL, USA).

# Results

## Characteristics of study population

*Table 1* shows the characteristics of the study population which comprised $n = 4429$ participants (34.9% male, median age 56.8 ± 9.8 years, range 19–87 years) of 6 ethnicities ($n = 3336$ Han, $n = 196$ Zhuang, $n = 225$ Uygur, $n = 285$ Kazak, $n = 193$ Dai, and $n = 194$ Korean) sampled

**Table 2 Blood pressure and heart rate measurements at baseline and 60 and 120 min following oral glucose (75 g)**

| Vital signs | Statistics | Fasting | 1 h | 2 h | P |
|---|---|---|---|---|---|
| SBP (mmHg) | *n* | 4429 | 4429 | 4312 | |
| | Mean ± SD | 135 ± 20 | 129 ± 19 | 127 ± 18 | <0.001 |
| | Difference between 1 h and fasting (95% CI)[a] | | | −6.2 (−6.6, −5.8) | |
| | Difference between 2 h and fasting (95% CI)[b] | | | −8.1 (−8.5, −7.7) | |
| DBP (mmHg) | *n* | 4429 | 4429 | 4312 | |
| | Mean ± SD | 84 ± 12 | 79 ± 11 | 78 ± 11 | <0.001 |
| | Difference between 1 h and fasting (95% CI)[a] | | | −4.7 (−4.9, −4.4) | |
| | Difference between 2 h and fasting (95% CI)[b] | | | −6.1 (−6.4, −5.8) | |
| HR (bpm) | *n* | 4412 | 4419 | 4305 | |
| | Mean ± SD | 73 ± 12 | 77 ± 12 | 76 ± 11 | <0.001 |
| | Difference between 1 h and fasting (95% CI)[a] | | | 4.3 (4.0, 4.5) | |
| | Difference between 2 h and fasting (95% CI)[b] | | | 2.6 (2.4, 2.9) | |
| SBP/DBP | ≥140/90 | 38.4% | 30.7% | 26.9% | <0.001 |
| | 120–140/80–90 | 41.8% | 38.7% | 39.4% | <0.001 |
| | <120/80 | 19.8% | 30.4% | 33.7% | <0.001 |

SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate.
[a]Calculated by 1 h minus fasting levels.
[b]Calculated by 2 h minus fasting levels.

from 6 provinces ($n = 2867$ Jiangsu, $n = 87$ Fujian, $n = 196$ Guangxi, $n = 809$ Xinjiang Uyghur autonomous region, ($n = 195$ Yunan, and $n = 275$ Jilin). Overall, 44.8% ($n = 1982$) participants had a previously diagnosed hypertension or high fasting BP recorded on the study day, 12.3% newly diagnosed diabetes, 21.4% dyslipidaemia, 0.6% chronic kidney disease, and 2.5% hepatic dysfunction. Compared with males, female participants were slightly younger, had lower UA levels, but comparable BMI, BP, and glycaemic and lipid profiles.

## Systolic blood pressure, diastolic blood pressure, and heart rate before and after a 75 g oral glucose load

The 75 g oral glucose load was associated with reductions in both SBP (by 6.2 [95% CI: 5.8, 6.6] mmHg and 8.1 [95% CI: 7.7, 8.5] mmHg at 1 and 2 h, respectively) and DBP (by 4.7 [95% CI: 4.4, 4.9] mmHg and 6.1 [95% CI: 5.8, 6.4] mmHg at 1 and 2 h, respectively), and an increase in HR (by 4.3 [95% CI 4.0, 4.5] bpm and 2.6 [95% CI: 2.4, 2.9] bpm at 1 and 2 h, respectively) ($P < 0.001$ for all) (*Table 2*, Supplementary material online, *Figure S2*).

The proportions of participants with high BP (i.e. ≥ 140/90 mmHg) were 38.4% when fasting ($n = 1700/4429$), 30.7% ($n = 1360/4429$) at 1 h, and 26.9% ($n = 1161/4312$) at 2 h after oral glucose. In 28.6% ($n = 1266/4429$) of individuals, BP was either high while fasting and normal at 2 h after the oral glucose load ($n = 872$), or was normal before and high after glucose ($n = 394$).

In males, high BP occurred in 38.6% when fasting, 31.7% at 1 h, and 28.0% at 2 h. In females, high BP occurred in 38.3% when fasting, 30.2% at 1 h, and 26.4% at 2 h (see Supplementary material online, *Table S1*).

In the subgroup with pre-existing hypertension, high BP occurred in 85.8% when fasting, 59.3% at 1 h, and 53.0% at 2 h (see Supplementary material online, *Table S1*).

In the subgroup with microalbuminuria, high BP occurred in 60.1% when fasting, 50.6% at 1 h, and 45.1% at 2 h. In the subgroup without microalbuminuria, high BP occurred in 33.3% when fasting, 26.0% at 1 h, and 22.6% at 2 h (see Supplementary material online, *Table S1*).

## Factors associated with changes in blood pressure and heart rate after oral glucose

After adjustment for potential confounders, including age, sex and ethnicity, changes from fasting SBP and DBP at 1 or 2 h were inversely associated with age, fasting SBP and DBP, hypertension, dyslipidaemia, and microalbuminuria, while changes in HR were greater in males than females, and associated directly with BMI and dyslipidaemia, but inversely with age, fasting BP, fasting HR, and hypertension (*Table 3*). Stratification of subjects according to age (below and above the median 58 years) yielded similar outcomes to those overall, with the exception of significant interactions between age, fasting SBP ($P = 0.038$), and hypertension ($P = 0.002$) in relation to the changes in DBP (see Supplementary material online, *Table S2*). However, the magnitude of the difference between subjects aged <58 years and ≥58 years was marginal. Stratification of subjects according to sex showed no interaction between sex and the aforementioned factors to influence the BP and HR responses to oral glucose (see Supplementary material online, *Table S3*). Stratification of subjects according to microalbuminuria status revealed a significant interaction between the presence of microalbuminuria and diabetes in relation to changes in SBP (coefficient −2.77, 95% CI: [−5.41, −0.12], $P = 0.040$), such that the reduction in SBP after oral glucose was greater among individuals with microalbuminuria and diabetes (see Supplementary material online, *Table S4*). Stratification of subjects according to a prior history of hypertension revealed significant interactions between hypertension and age (coefficient −0.06, 95% CI: [−0.10, −0.01], $P = 0.010$) and dyslipidaemia (coefficient −1.10, 95% CI: [−2.15, −0.06], $P = 0.039$) in relation to changes in DBP. However, the magnitude of differences between subgroups with and without a prior history of hypertension were marginal (see Supplementary material online, *Table S5*). Association analyses of the changes in SBP and HR at 1 and 2 h after oral glucose also revealed

**Table 3 Factors associated with changes in blood pressure and heart rate following oral glucose (75 g)[a]**

| Characteristics | Coefficient (95% CI), *P*-value | | |
|---|---|---|---|
| | ΔSBP | ΔDBP | ΔHR |
| Sex (male) | 0.66 (−0.08, 1.40), 0.081 | −0.27 (−0.74, 0.20), 0.261 | 0.77 (0.25, 1.30), 0.004 |
| Age (year) | −0.09 (−0.13, −0.06), < 0.001 | −0.04 (−0.07, −0.02), < 0.001 | −0.04 (−0.07, −0.01), 0.003 |
| BMI (kg/m$^2$) | −0.02 (−0.12, 0.07), 0.631 | −0.03 (−0.09, 0.03), 0.376 | 0.11 (0.04, 0.17), 0.002 |
| Fasting SBP | −0.30 (−0.31, −0.28), < 0.001 | −0.07 (−0.08, −0.06), < 0.001 | −0.03 (−0.04, −0.01), < 0.001 |
| Fasting DBP | −0.20 (−0.23, −0.18), < 0.001 | −0.30 (−0.32, −0.29), < 0.001 | −0.04 (−0.06, −0.02), < 0.001 |
| Fasting HR | −0.02 (−0.05, 0.01), 0.146 | −0.04 (−0.06, −0.02), < 0.001 | −0.33 (−0.35, −0.31), < 0.001 |
| Hypertension | −8.51 (−9.18, −7.85), < 0.001 | −4.23 (−4.66, −3.80), < 0.001 | −0.87 (−1.38, −0.36), 0.001 |
| Diabetes | 1.09 (−0.07, 2.26), 0.065 | 0.35 (−0.39, 1.09), 0.357 | −0.55 (−1.38, 0.27), 0.190 |
| Dyslipidaemia | −0.97 (−1.83, −0.11), 0.027 | −0.63 (−1.18, −0.09), 0.022 | 0.50 (−0.11, 1.11), 0.107 |
| Chronic kidney disease | −2.89 (−7.53, 1.74), 0.222 | 0.98 (−1.96, 3.91), 0.513 | −2.14 (−5.44, 1.15), 0.202 |
| Microalbuminuria | −2.08 (−3.00, −1.16), < 0.001 | −0.95 (−1.53, −0.37), 0.001 | −0.51 (−1.16, 0.15), 0.130 |
| Hepatic dysfunction | 1.72 (−0.54, 3.99), 0.136 | 1.42 (−0.02, 2.85), 0.052 | 0.51 (−1.10, 2.13), 0.533 |

SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; BMI, body mass index.
Dyslipidaemia: Elevated TC, TG, LDL-c, or decreased HDL-c.
Chronic kidney disease: Decreased eGFR (lower than 60).
Hepatic dysfunction: Elevated ALT, AST, GGT, or Tbil (2 times higher than upper limit).
Microalbuminuria: Urine albumin to creatinine ratio over 30 mg/g.
[a]Adjusted for age, sex, and ethnicity.

significant correlations between the two parameters in participants without PPH (1 h: $r = 0.097$, $P < 0.001$; 2 h: $r = 0.090$, $P < 0.001$), but not in those with PPH (1 h: $r = 0.042$, $P = 0.165$; 2 h: $r = 0.059$, $P = 0.053$).

## Prevalence of postprandial hypotension and its association with macro- and micro-vascular disease

The overall prevalence of PPH in the was 24.9% ($n = 1101/4429$). After adjustment for age and sex distribution according to the 2020 Chinese population census data, the estimated prevalence of PPH in the general Chinese adult population was 19.9% (95% CI: 18.7, 21.1%), with a higher prevalence in females than males (21.8% [95% CI: 20.3, 23.3%] vs. 18.0% [95% CI: 16.1, 20.0%], $P = 0.003$) and an increasing prevalence with ageing (from 12.5% [95% CI: 8.6, 16.3%] in participants <40 years to 27.6% [95% CI: 22.3, 32.8%] in those ≥ 70 years, $P < 0.001$) (*Table 4*). The prevalence of PPH was higher in overweight and obese than lean participants (26.2% [95% CI: 23.4, 29.1%] in obese, 26.2% [95% CI: 24.2, 28.3%] in overweight, and 22.9% [95% CI: 20.9, 24.8%] in lean participants, $P = 0.04$), and in those with hypertension (39.8% [95% CI: 37.6, 31.9%] vs. 12.8% [95% CI: 11.5, 14.1%], $P < 0.001$) and prediabetes (normal glucose tolerance 23.3% [95% CI: 21.8, 24.9%], prediabetes 29.2% [95% CI: 26.7, 32.0%], diabetes 23.7% [95% CI: 19.8, 27.6%], $P < 0.001$) than those without (*Table 4*). Stratification of participants according to fasting SBP, cigarette smoking, alcohol intake, and the use of anti-hypertensive medications further showed that the prevalence of PPH was higher among individuals with higher fasting SBP, cigarette smoking, and alcohol intake, but was not affected by the use of antihypertensive medications (see Supplementary material online, *Table S6*).

After adjustment for age, sex, ethnicity, hypertension, glycaemic state, CKD, and microalbuminuria, female sex (OR 1.26, 95% CI 1.08, 1.47), age (per 5-year increment, OR 1.05, 95% CI 1.01, 1.10), hypertension (OR 4.55, 95% CI 3.89, 5.33), and CKD (OR 2.88, 95% CI 1.19, 6.97) were identified as independent risk factors for PPH. Surprisingly, diabetes appeared to be associated with a reduced risk of PPH (OR 0.72, 95% CI 0.56, 0.93) (*Table 5*).

The prevalence of CVD, stroke, and combined macrovascular disease were substantially higher in those with PPH than those without (CVD 6.3% vs. 3.9%, stroke 3.1% vs. 1.7%, combined 9.2% vs. 5.1%, all $P < 0.05$); after adjustment for age, sex, ethnicity, BMI, hypertension, HbA1c, TC, TG, and eGFR, PPH was associated with a higher risk of combined CVD and stroke (OR 1.49, 95% CI 1.13–1.97) (*Table 6*). While the prevalence of retinopathy and microalbuminuria were also higher in participants with PPH, after adjustment for sociodemographic and metabolic confounders, PPH was no longer associated with microvascular disease.

# Discussion

This large, nationwide study, involving community-dwelling adult participants from different geographic regions and ethnic backgrounds in China, has demonstrated a high prevalence of a marked fall in BP following a 75 g oral glucose load. This phenomenon would have a significant impact on the diagnosis of hypertension, given that about a third of individuals with an abnormally high BP level at baseline were normotensive during the 2 h after the glucose drink. Moreover, about 25% of the study population (equating to ∼20% of the standardized Chinese adult population) exhibited a fall in SBP of greater than 20 mmHg from baseline after oral glucose, meeting the widely used diagnostic criterion for PPH. The latter was associated with both increasing age and hypertension, and also predictive of a marked increase in macrovascular disease. These observations are indicative of major limitations in current practice relating to BP measurement and interpretation of BP outcomes.

Current guidelines for BP measurement generally lack information about the impact of meal ingestion, although some recommend that

**Table 4 Prevalence of postprandial hypotension stratified by demographic characteristics and health status**

| Characteristic | No of participants[a] | No of patients with PPH[a] | Prevalence (%) | *P* value[b] |
|---|---|---|---|---|
| Overall | 4429 | 1101 | 19.9 (18.7, 21.1)[c] | |
| Sex | | | | |
| Male | 1547 | 362 | 18.0 (16.1, 20.0)[c] | 0.003 |
| Female | 2882 | 739 | 21.8 (20.3, 23.3)[c] | |
| Age (years) | | | | |
| 19–39 | 289 | 39 | 12.5 (8.6, 16.3)[c] | <0.001 |
| 40–49 | 602 | 130 | 21.4 (18.1, 24.7)[c] | |
| 50–59 | 1710 | 443 | 25.3 (23.2, 27.3)[c] | |
| 60–69 | 1545 | 411 | 26.5 (24.3, 28.7)[c] | |
| ≥70 | 283 | 78 | 27.6 (22.3, 32.8)[c] | |
| BMI (kg/m$^2$) | | | | |
| <24.0 | 1750 | 400 | 22.9 (20.9, 24.8) | 0.040 |
| 24.0–27.9 | 1758 | 461 | 26.2 (24.2, 28.3) | |
| ≥28.0 | 903 | 237 | 26.2 (23.4, 29.1) | |
| Hypertension | | | | |
| No | 2447 | 313 | 12.8 (11.5, 14.1) | <0.001 |
| Yes | 1982 | 788 | 39.8 (37.6, 41.9) | |
| Glycaemic status | | | | |
| Normal glucose tolerance | 2848 | 664 | 23.3 (21.8, 24.9) | <0.001 |
| Prediabetes | 1125 | 329 | 29.2 (26.6, 31.9) | |
| Diabetes | 456 | 108 | 23.7 (19.8, 27.6) | |

[a]Unweighted number of participants and PPH cases.
[b]Prevalence between groups were compared using $\chi^2$ test. Data are presented as percentage and 95% CI.
[c]Estimated prevalence of PPH in the general population, adjusted for age and/or sex of the latest Chinese population census data.

**Table 5 Factors associated with postprandial hypotension[a]**

| Characteristics | Odds ratio (95% CI), *P*-value |
|---|---|
| Female | 1.26 (1.08, 1.47), 0.004 |
| Age (per 5 year increment) | 1.05 (1.01, 1.10), 0.011 |
| Hypertension | 4.55 (3.89, 5.33), <0.001 |
| Glycaemic state | |
| Normal glucose tolerance | Reference |
| Prediabetes | 1.06 (0.90, 1.26), 0.478 |
| Diabetes | 0.72 (0.56, 0.93), <0.001 |
| Chronic kidney disease | 2.88 (1.19, 6.97), 0.019 |
| Microalbuminuria | 0.97 (0.81, 1.17), 0.974 |

[a]Logistic regression model adjusted for age, sex, ethnicity, hypertension, glycemic state, CKD, and microalbuminuria

**Table 6 Association of postprandial hypotension with comorbidities**

| Vascular disease | Prevalence in non-PPH (%) | Prevalence in PPH (%) | Adjusted odds ratio (95% CI) of PPH[a] |
|---|---|---|---|
| Macrovascular disease | 171/3325 (5.1) | 99/1076 (9.2)[b] | 1.49 (1.13, 1.97) |
| CVD | 129/3325 (3.9) | 68/1072 (6.3)[b] | 1.31 (0.95, 1.82) |
| Stroke | 57/3325 (1.7) | 33/1074 (3.1)[b] | 1.51 (0.95, 2.39) |
| Micro-vascular disease | 693/3321 (20.9) | 293/1099 (26.7)[b] | 0.96 (0.81, 1.14) |
| Retinopathy | 192/3211 (6.0) | 65/1064 (6.1) | 0.87 (0.64, 1.19) |
| Microalbuminuria | 550/3169 (17.4) | 253/1070 (23.6)[b] | 0.98 (0.81, 1.17) |

[a]Multivariable logistic regression adjusted for age, sex, ethnicity, body mass index (BMI), hypertension, glycated haemoglobin (HbA1c), total cholesterol (TC), triglycerides (TG), and estimated globular filtration rate (eGFR).
[b]$P < 0.05$ compared with the prevalence in non-PPH using $\chi^2$ test.

BP is measured before breakfast and ingestion of medications for home BP monitoring.[4,6,8,9] Our study establishes that there is a major discrepancy in the detection of an abnormally high BP among males and females, individuals with a prior history of hypertension, and those with and without microalbuminuria, depending on whether BP is measured before or after oral glucose, reflecting the hypotensive response to the latter.

We have shown previously that the rate of gastric emptying, which varies substantially even between healthy individuals in the range of 1–4 kcal/min,[28] is a major determinant of the hypotensive response to oral glucose.[29] Although a glucose drink differs from a physiological meal, previous work has shown that carbohydrate, fat, and proteins have comparable effects to reduce BP.[30,31] Moreover, the rate of gastric emptying, which is a dominant determinant of the BP response to a meal[32] is dependent on caloric content, rather than macronutrient composition.[30] Accordingly, we believe that our findings are of relevance to the ingestion of physiological meals so that measurement of

BP should incorporate standardization of meal ingestion, involving both fasting and postprandial measurements—otherwise there is a high risk of misinterpreting BP results measured at convenience. For example, individuals who had abnormally high BP during fasting may be normotensive or even hypotensive, after a meal. Furthermore, the fasting and postprandial phases have not been discriminated in relation to the effect of antihypertensive drugs. We acknowledge that the diagnostic cut-off for PPH is somewhat arbitrary, attesting to the relative lack of clinical attention to this condition. While the current study was not designed to address this issue, we found that PPH diagnosed by a fall in SBP of ≥20 mmHg after a 75 g oral glucose load was associated with increased vascular comorbidities. Moreover, that the use of a glucose drink for the detection of PPH can be readily combined with measurement of plasma glucose in screening for diabetes, adds to the diagnostic value.

After nutrient intake, blood is redistributed to the gut leading to compensatory increases in HR and systemic resistance, which are governed by autonomic function. When cardiovascular compensation is inadequate, BP falls. Indeed, in participants without PPH, the changes in SBP at 1 and 2 h after oral glucose correlated with the corresponding changes in HR, but these correlations were not evident in participants with PPH. As in previous studies,[14,15,33] we found that age and fasting BP were positively associated with the decline in postprandial BP, and negatively associated with the increase in HR, implying that in older individuals and those with hypertension the rise in HR was insufficient to maintain BP at fasting levels. The mechanisms underlying these associations remain to be defined, but may relate, at least in part, to autonomic dysfunction, which we did not assess, and the attenuated gastrovascular reflex seen in the elderly and in those with hypertension.[34] We recognize that vagal stimulation can result from glucose ingestion and may potentially drive a fall in BP, but this is typically accompanied by a concurrent fall in HR, rather than the observed increase.[35] Accordingly, vagal stimulation is most unlikely to account for the fall in BP evident in our study.

Based on the outcomes of our study, it can be estimated that about 20% of the total Chinese adult population would have PPH after a 75 g oral glucose load. Previous studies in relatively small samples indicated that PPH occurs commonly in the elderly, those with hypertension, and in individuals with long-standing type 1 or type 2 diabetes,[14,17,32,36] but the current study is the first to show that PPH occurs frequently in the general adult population. We also found that PPH was associated with a 51% increase in the combined risk of CVD and stroke, even after adjusting for age, hypertension, HbA1c and lipid levels. Our observations extend insights derived from prospective cohort studies in low-level care residents and older nursing home residents, which indicate that PPH represents an independent risk factor for the onset of coronary events, stroke, and overall mortality,[15,33] and a recently published meta-analysis which showed that PPH was associated with increased total CVD and stroke, as well as all-cause mortality and CVD mortality.[18] Accordingly, PPH is likely to represent an important target for preventing and managing macrovascular and micro-vascular disease and warrants evaluation in prospective studies. The fact that management options for PPH are currently limited, and suboptimal, must also be addressed.

In interpreting our findings, several limitations should be noted. First, females and older individuals were oversampled, as males and younger residents were often working outside their permanent residential regions. To account for this, we adjusted the estimated prevalence of PPH in the general community for the distribution of sex and age according to the 2020 national population census. Moreover, the observed prevalence of diabetes and hypertension were comparable to those reported in other large-scale population-based studies (diabetes 12.8%, hypertension 44.7%),[37,38] supporting the representative nature of our study sample. It should be noted that people with known diabetes were excluded from the study and diabetes represented an incidental, rather than established, diagnosis. It was, accordingly, to be expected that the number of participants with diabetes was more modest when compared to those with normoglycaemia or prediabetes. The apparently reduced risk of PPH in the context of diabetes was surprising and likely to represent the outcome of these confounders. Second, individuals with known diabetes, mental illness, and other disorders that would prevent them from completing the study procedures, as well as those who were pregnant, were excluded, with the inherent potential for selection bias. Third, BP was not measured continuously for logistical reasons, potentially leading to an under-reporting of the prevalence of PPH. While it is unclear whether the falls in BP would be more marked after lunch and evening meals, there is evidence that the fall is usually greatest after breakfast,[39] and variations in BP after breakfast are more closely associated with cardiovascular mortality.[17] Twenty-four-hour ambulatory BP monitoring would be ideal for evaluating overall postprandial BP changes and for the detection of PPH but was not feasible in a study of this scale. Similarly, for logistical reasons, BP measurements were only performed on the study day, rather than on three different days, as suggested by current guidelines. This would affect the accuracy of the diagnosis of hypertension. Furthermore, for logistical reasons, BP was only measured once at each time point, which would inevitably increase data variability. Participants were instructed to withhold their antihypertensive medications only the night before or on the morning of the study, as we considered that more prolonged withholding of these medications would be unethical. This approach may, however, have accounted for the rise in BP in a small subset of participants. Fourth, our study lacked a control group who drank only water, so a gradual reduction in anxiety, rather than glucose intake, may potentially have affected BP levels. However, in previous studies which used water as a control, BP levels were still lower after oral glucose.[40] Importantly, the reduction in BP after oral glucose was accompanied by persistent increases in HR. Accordingly, it is highly unlikely that the fall in BP was driven by 'resting' or ease of anxiety. We recognise that the resting phase before each BP measurement, albeit complying with many guidelines,[4,8–10] was relatively brief, but prolongation of the resting phase would have compromised the feasibility of this study. While there may have been inaccuracy in BP measurements generally, this would have led to random data misclassification—that is, not biased towards any subgroup of interest—and, hence, conservative estimates of differences among them. Future studies, involving a non-nutritive drink as a control, would add support on the impact of a nutrient load on BP levels. Fifth, this was a cross-sectional study with self-reported macrovascular diseases, so that reporting bias cannot be excluded. Since evaluation of BP and HR responses was conducted along with screening of diabetes, diabetic micro-vascular complications, including diabetic retinopathy and diabetic kidney disease, were used as a surrogate of micro-vascular events. It should also be noted that in our study, the potential determinants of changes in BP and HR were exploratory, and could be compromised by inadequate adjustment for other unknown confounders. Longitudinal studies are needed to confirm the consequences of PPH in the general adult population. Finally, the current study was conducted only in a Chinese adult population. In view of substantial ethnic disparities in the regulation of metabolic homeostasis, further studies to evaluate the prevalence and consequences of PPH in other populations are warranted.

# Conclusion

In conclusion, our study showed that there is a substantial decrease in BP after oral glucose in a Chinese community-dwelling adult population, with 20% meeting the criteria for PPH. Our observations highlight the need for standardization of BP measurement in relation to meals, and indicate that PPH, in addition to hypertension, represents a major target for clinical intervention to prevent CVD and stroke.

# Supplementary material

Supplementary material is available at *European Journal of Preventive Cardiology*.

# Author contributions

Z.S., K.L.J., S.Q., B.W., M.H., C.K.R., T.W., D.L., and D.W. conceived and designed the study. B.W., H.G., K.L., Q.W., X.W., Q.C., H.L., S.Y., X.Z., and M.S. conducted the epidemiological survey. X.Z., M.S., S.Q., and DW performed the statistical analysis. All authors contributed to acquisition, analysis, or interpretation of data. X.Z. and T.W. drafted the manuscript. All authors revised the report and approved the final version before submission.

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# Ethical approval and consent to participate

The study was approved by the Human Research Ethics Committee of Zhongda Hospital, Southeast University, Nanjing, China. All participants provided written informed consent.

**Conflict of interest:** M.H. has participated in the advisory boards and/or symposia for Novo Nordisk, Sanofi, Novartis, Eli Lilly, Merck Sharp & Dohme, Boehringer Ingelheim, and AstraZeneca and has received honoraria for this activity. C.K.R. has received research funding from AstraZeneca, Merck Sharp & Dohme, Eli Lilly, Novartis, and Sanofi. T.W. has received travel support from Novartis and Sanofi and research funding from Novartis and AstraZeneca. None of the other authors has any personal or financial conflict of interest to declare.

## Data availability

Relevant individual de-identified participant data will be made available on reasonable request via e-mail to Z.S.

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