

Association between blood pressure fluctuation and left ventricular remodeling and the mediation role of chronic low-grade inflammation in older adults: A community-based cross-sectional study

Zhendong Liu

zhendongliu876@126.com

Institute of Basic Medicine, Shandong Academy of Medical Sciences

Xiaojing Zhang

Jia Liu

Peilin Li

Siyi Dun

Chunju Zhang

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Abstract

We aimed to investigate the correlation between blood pressure (BP) fluctuation, including BP variability (BPV) and circadian rhythm, and left ventricular remodeling (LVR), and to explore the potential mediating role of chronic low-grade inflammation in these correlations. Between May 2021 and October 2023, 548 community-dwelling adults aged ≥ 60 years were assessed: BPV and circadian rhythm were evaluated using 24-hour ambulatory BP monitoring, LVR was assessed by echocardiography, and inflammation was measured via hypersensitive C-reactive protein (hsCRP), tumor necrosis factor- α (TNF- α), and monocyte chemotactic protein-1 (MCP-1). Twenty-four-hour systolic (S)BP standard deviation (SD) and coefficient of variation (CV) were showed to be positively correlated with interventricular septal thickness, left ventricular posterior wall thickness, and left ventricular mass index, while SBP and diastolic (D)BP nocturnal dipping rates (NDR) were negatively correlated with these parameters. Left ventricular hypertrophy (LVH) risk showed positive linear dose-response correlations with 24-hour SBP SD and CV, an L-shaped correlation with SBP NDR, and a negative linear correlation with DBP NDR. After adjustment for confounders including 24-hour average SBP and DBP, compared with dipper rhythm, LVH risk was elevated by 0.874-fold (OR: 1.874, 95% CI: 1.113–3.155) in non-dippers and by 3.965-fold (OR: 4.965, 95% CI: 2.847–8.659) in reverse dippers. HsCRP, TNF- α , and MCP-1 collectively mediated the correlation between LVR and BP fluctuation. In conclusion, excessive BPV and abnormal circadian BP rhythms, especially reverse dipper rhythms, are risk factors for LVR in older adults, and chronic low-grade inflammation plays a crucial mediating role in these correlations.

Trial registration: Registered number: ChiCTR1900023580

Introduction

Left ventricular remodeling (LVR) refers to structural changes in the heart that impair systolic and diastolic function, which is the main cause of myocardial infarction and heart failure [1, 2]. The association between LVR and elevated blood pressure (BP) has been well established. Strictly lowering BP therapy is widely recommended by several guidelines for hypertension management [3–5]. Yet, fluctuation is an inherent characteristic of BP. Evidence demonstrated that BP fluctuation, including BP variability (BPV) and circadian rhythm, is increased with BP elevating but also is not significantly affected by BP lowering medications [6, 7].

In addition to elevated average BP levels, excessive BPV and abnormal circadian BP rhythm has been revealed to be associated with several chronic conditions [8–11]. Song and colleagues demonstrated the excessive visit-to-visit BPV is associated with the progression of atherosclerosis and endothelial dysfunction in hypertensive patients [8]. Zhang H et al. demonstrated that abnormal BP rhythms such as non-dippers and reverse dippers are significantly associated with an increased risk of cerebral small vessel diseases [9]. Therefore, we propose the hypothesis that, in addition to elevated BP levels, excessive BPV and abnormal circadian BP rhythm both constitute additional crucial risk factors for LVR. However, evidence regarding the relationship between LVR and BP fluctuations remains limited.

Ambulatory BP monitoring (ABPM) is the gold standard for assessing average 24-hour BP levels and fluctuations. It is widely recommended by major guidelines for evaluating BP variability [3, 12, 13]. Unlike office BP, home self-measured BP, and visit-to-visit BP measurements, ABPM can assess BP variability within 24 hours and circadian rhythm patterns. Importantly, the BP fluctuations assessed using ABPM are minimally impacted by antihypertensive medication use [14].

Chronic low-grade inflammation plays a vital role in the occurrence and progression of cardiovascular diseases [15, 16]. Hypersensitive C-reactive protein (hsCRP), tumor necrosis factor α (TNF- α), and monocyte chemotactic protein-1 (MCP-1) are classical markers of chronic inflammation. Kim and co-workers demonstrated that hsCRP and TNF- α are linked to elevated BPV [17]. Li and colleagues found that hsCRP and TNF- α are closely associated with morning hypertension and atrial fibrillation [6]. Although low-grade inflammation has been revealed to be associated with LVR and hypertension, the role of inflammation in the association between LVR and BP fluctuation is seldom reported. In this study, we employed hsCRP, TNF- α , and MCP-1 to comprehensively assess the inflammatory levels and to elucidate their mediating role in the association between LVR and BP fluctuation.

In this study, our main goals were to investigate the association between LVR and BP fluctuation and the mediating role of chronic low-grade inflammation on this association in a community-dwelling older adults.

Materials and methods

Study population:

This study was derived from a community-based observational investigation (Registration number: ChiCTR1900023580), which aimed to elucidate the prevention, treatment, and rehabilitation of chronic cardiovascular and cerebrovascular diseases. Between May 2021 and October 2023, a total of 548 older adults, aged 60 years and above, were recruited from the Shandong region of China. The exclusion criteria were as follows: absence ABPM, echocardiography, or chronic inflammation assessment, pacemaker use, congestive heart failure, myocardial infarction, stroke, acute or chronic infectious diseases, immunosuppressant use, liver or renal dysfunction, malignancy, drug or alcohol abuse, and unwillingness to provide informed consent.

This study was conducted in compliance with the Declaration of Helsinki and adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines. The study was approved by the Research Ethics Committee of the Institute of Basic Medicine, Shandong

BP fluctuation assessment and classification:

Noninvasive 24-hour ABPM was performed using validated oscillometric devices (TM-2430; A&D Co. Inc., Tokyo, Japan). BP and pulse rate were recorded every 15 min during daytime (0600–2200) and every 20 min during nighttime (2200–0600) [ref. 6,9]. The valid recordings required $\geq 70\%$ of daytime or nighttime measurements, ≥ 20 valid readings with at least two valid readings per hour while awake, and ≥ 7 valid readings with at least one valid reading per hour while asleep [6, 9]. BP profiles were assessed using the average of within-subject readings. BPV was calculated as the standard deviation (SD) and coefficient of variation (CV) in serial BP measurements. The calculated formula of CV was: $CV (\%) = SD \text{ of BP} / \text{mean of BP} \times 100\%$ [ref. 17]. Circadian BP rhythm were identified using nocturnal dipping rate (NDR) of 24-hour systolic (S)BP. NDR was calculated with the formula: $NDR (\%) = (1 - \text{average nighttime SBP} / \text{average daytime SBP}) \times 100\%$. Four circadian BP rhythms were identified: Extreme dippers was defined as $NDR \geq 20\%$, dippers as $10\% \leq NDR < 20\%$, non-dippers as $0\% \leq NDR < 10\%$, and reverse dippers as $NDR < 0\%$ [ref. 9].

Echocardiography

Echocardiography was performed by experienced echocardiographers, who blinded to clinical data, using a high-resolution ultrasound system (Vivid 7, GE Medical Systems Ultrasound Israel Ltd, Israel) [18, 19]. Interventricular septal thickness (IVST), left ventricular posterior wall thickness (LVPW), left ventricular end-diastolic internal dimension (LVDD), were measured on the edges of mitral leaflets by cutting the parasternal long axis vertically with M-mode [18–21]. Left ventricular mass (LVM) was calculated as follows: $LVM (g) = 0.8 \times 1.04 \times [(IVST + LVDD + LVPW)^3 - (LVDD)^3] + 0.6$ [ref. 19]. LVM index (LVMI) was computed with the formula: $LVMI (g/m^2) = LVM (g) / \text{body surface area (m}^2\text{)}$ [ref. 19]. Basing on the American Society of Echocardiography and the European Association of Cardiovascular Imaging, left ventricular hypertrophy (LVH) was defined as an $LVMI > 95 g/m^2$ in women and $> 115 g/m^2$ in men [20]. Accordingly, participants were classified into the LVH and non-LVH groups.

Inflammation assessment:

Intravenous blood samples were obtained from each participant in the morning after overnight fasting. The plasma levels of hsCRP, TNF- α , and MCP-1 were detected using enzyme-linked immunosorbent assay kits (Beyotime, Shanghai, China) according to the manufacturer's instructions [6].

Covariates:

The covariates included age, sex, smoking, alcohol consumption, regular exercise, histories of hypertension, diabetes, and dyslipidemia, histories of anti-hypertensive, anti-diabetic, and anti-dyslipidemic medication use, body mass index (BMI), pulse, total cholesterol (TCHO), triglyceride (TG), high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), fasting plasma glucose (FPG).

Statistic analyses:

Continuous data were expressed as mean $\pm$ SD or median with interquartile range (IQR; 25th and 75th percentiles) based on the normality assessed using the Kolmogorov-Smirnov test. Categorical data were presented as frequencies and percentages. The independent samples t-test was employed to determine differences in normalized continuous data, while the Mann-Whitney U-test was used to assess differences in non-normalized continuous data. The Chi-square test was conducted to evaluate differences in categorical data. Pearson's or Spearman's correlation analyses were performed to assess correlations between the measurements of BPV, BP NDR, LVR, and inflammation when appropriate. Subsequently, a multiple linear backward stepwise regression analysis was applied to determine the independent associations between these variables. Models were adjusted for the confounders including: age, sex, smoking, alcohol consumption, regular exercise, histories of hypertension, dyslipidemia, and diabetes, and corresponding medication use, BMI, office SBP and DBP, TCHO, TG, HDL-c, LDL-c, and FPG.

Restricted cubic splines (RCS) analytic model was used to determine the dose-response correlations of LVH to 24-hour SBP and DBP SD and CV, SBP and DBP NDR, and inflammation profiles. Logistic regression analysis was conducted to evaluate the odds ratio (OR) with 95% confidence intervals (CI) in participants with extreme dippers, non-dippers, and reverse dippers compared to those with dippers. SPSS Hayes PROCESS (version 4.0) was conducted to assess the mediating role of hsCRP, TNF- α , and MCP-1 in the associations between profiles of LVR and profiles of BP fluctuation. The variance inflation factor (VIF) for all covariates was evaluated. The VIF for all covariates in this study was < 5 , and if the VIF was ≥ 5 , the corresponding covariate was excluded from the analysis model aimed to control for result bias caused by multicollinearity.

All statistical analyses were performed using SPSS for Windows software package (version 26.0, SPSS Inc.), and graphs were generated with GraphPad Prism (version 9.1.0, GraphPad Software) and Adobe Illustrator (version 2024, Adobe). A two-sided $p < 0.05$ was considered statistically significant.

Results

Baseline demographic and clinical characteristics of participants

Among the 548 participants, 298 (54.4%) were female, 396 (72.3%) had hypertension, 92 (16.8%) had diabetes, 185 (33.9%) smoked, and 205 (37.4%) consumed alcohol. The mean age was 67.58 (SD: 5.69) years, and the average LVMI was 100.12 (IQR: 81.23 to 126.78) g/m². There were 204 (37.2%) participants with LVH, and 344 (62.8%) participants with non-LVH. Table 1 detailed the participants' baseline clinical characteristics and Fig. 1 presented the study flowchart.

Table 1
Baseline demographic and clinical characteristics

	Total Participants (<i>n</i> = 548)	Classified by LVR		<i>p</i> value
		LVH (<i>n</i> = 204)	Non-LVH (<i>n</i> = 344)	
Age, years	67.58 ± 5.69	68.03 ± 5.83	67.31 ± 5.60	0.151
Female, <i>n</i> (%)	298 (54.4)	107 (52.5)	191 (55.5)	0.485
Hypertension, <i>n</i> (%)	396 (72.3)	165 (80.9)	231 (67.2)	< 0.001
Dyslipidemia, <i>n</i> (%)	193 (35.2)	67 (32.8)	126 (36.6)	0.370
Diabetes, <i>n</i> (%)	92 (16.8)	40 (19.6)	52 (15.1)	0.174
Smoking, <i>n</i> (%)	186 (33.9)	65 (31.9)	121 (35.2)	0.429
Alcohol consumption, <i>n</i> (%)	205 (37.4)	77 (37.7)	128 (37.2)	0.900
Regular exercise, <i>n</i> (%)	307 (56.0)	104 (51.0)	203 (59.0)	0.067
Anti-hypertensive medication use, <i>n</i> (%)	276 (50.4)	107 (52.5)	169 (49.1)	0.452
Anti-dyslipidemic medication use, <i>n</i> (%)	101 (18.4)	37 (18.1)	64 (18.6)	0.891
Anti-diabetic medication use, <i>n</i> (%)	83 (15.1)	36 (17.6)	47 (13.7)	0.209
BMI, kg/m ²	24.9 ± 2.1	25.24 ± 2.12	24.64 ± 2.12	0.688
Office SBP, mmHg	147.6 ± 14.9	149.55 ± 14.77	146.36 ± 14.92	0.681
Office DBP, mmHg	76.9 ± 8.4	77.33 ± 8.29	76.66 ± 8.46	0.525
TCHO, mmol/L	4.70 ± 0.68	4.76 ± 0.66	4.67 ± 0.69	0.128
TG, mmol/L	1.51 (1.15, 1.88)	1.52 (1.19, 1.86)	1.49 (1.11, 1.88)	0.740
HDL-c, mmol/L	1.07 (0.92, 1.39)	1.04 (0.80, 1.29)	1.09 (0.86, 1.32)	0.120
LDL-c, mmol/L	2.80 (2.36, 3.25)	2.87 (2.49, 3.25)	2.73 (2.26, 3.21)	0.032
FPG, mmol/L	5.17 (4.72, 6.00)	5.42 (4.75, 6.09)	5.08 (4.50, 5.67)	< 0.001
Profiles of 24-hour ABPM				
24-hour SBP, mmHg	135.00 (124.00, 148.00)	140.50 (130.00, 150.00)	133.00 (122.00, 145.00)	< 0.001
24-hour average DBP, mmHg	71.00 (65.25, 78.00)	72.00 (66.25, 78.00)	71.00 (65.00, 78.00)	0.343
Daytime average SBP, mmHg	138.00 (128.00, 149.00)	141.00 (131.00,151.00)	136.50 (128.00, 148.00)	0.042
Daytime average DBP, mmHg	72.00 (66.00, 78.00)	73.00 (67.00, 78.00)	72.00 (66.00, 78.00)	0.457
Nighttime average SBP, mmHg	128.00 (114.00, 142.00)	137.50 (123.00, 148.75)	124.00 (111.00, 139.00)	< 0.001
Nighttime average DBP, mmHg	70.00 (63.00, 76.00)	72.00 (65.00, 77.75)	69.00 (63.00, 76.00)	0.010
24-hour SBP SD, mmHg	35.51 (29.61, 42.82)	40.09 (33.14, 46.92)	33.06 (27.53, 39.83)	< 0.001
24-hour DBP SD, mmHg	13.88 (11.57, 16.38)	14.00 (11.67, 16.46)	13.81 (11.51, 16.38)	0.516
24-hour SBP CV, %	26.28 (21.83, 31.65)	28.77 (24.02, 33.58)	25.26 (21.07, 29.40)	< 0.001
24-hour DBP CV, %	19.88 (16.61, 22.85)	19.93 (17.07, 22.85)	19.70 (16.58, 22.85)	0.610
SBP NDR, %	6.20 (-0.87, 13.60)	0.73 (-3.48, 8.45)	9.24 (2.60, 16.25)	< 0.001
DBP NDR, %	3.57 (0.00, 6.25)	2.33 (0.00, 5.00)	4.26 (1.45, 6.75)	< 0.001
BP circadian patterns				< 0.001
The results are presented as the mean ± SD, median with interquartile range, or frequencies and percentages. LVR, left ventricular remodeling; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TCHO, total cholesterol; TG, triglyceride; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; FPG, fasting plasma glucose; SD, standard deviation; CV, coefficient of variance; NDR, nocturnal dipping rate; IVST, interventricular septal thickness; LVPW, left ventricular posterior wall thickness; LVDD, left ventricular end-diastolic internal dimension; LVM, left ventricular mass; LVMI, left ventricular mass index; LVH, left ventricular hypertrophy; hsCRP, hypersensitive C-reactive protein; TNF-α, tumor necrosis factor α; MCP-1, monocyte chemotactic protein-1.				

	Total Participants (<i>n</i> = 548)	Classified by LVR		<i>p</i> value
		LVH (<i>n</i> = 204)	Non-LVH (<i>n</i> = 344)	
Dippers, <i>n</i> (%)	144 (26.3)	32 (15.7)	112 (32.6)	
Extreme dippers, <i>n</i> (%)	63 (11.5)	12 (5.9)	51 (14.8)	
Non-dippers, <i>n</i> (%)	187 (34.1)	67 (32.8)	120 (34.9)	
Reverse dippers, <i>n</i> (%)	154 (28.1)	93 (45.6)	61 (17.7)	
Profiles of LVR				
IVST, mm	10.80 (9.74, 12.09)	12.40 (11.79, 13.07)	10.20 (9.25, 10.86)	< 0.001
LVPW, mm	10.90 (9.67, 12.24)	11.95 (11.00, 13.08)	10.12 (9.15, 11.42)	< 0.001
LVDD, mm	45.35 ± 4.99	47.27 ± 5.30	44.21 ± 4.42	< 0.001
LVM, g	178.32 ± 46.41	218.55 ± 40.56	154.46 ± 30.47	< 0.001
LVMI, g/m ²	100.12 (81.23, 126.78)	129.80 (123.91, 142.12)	87.62 (74.05, 100.17)	< 0.001
Inflammation markers				
HsCRP, mg/dL	0.60 (0.41, 0.80)	0.77 (0.58, 0.91)	0.52 (0.38, 0.70)	< 0.001
TNF-α, pg/mL	61.64 (45.80, 77.30)	72.93 (58.50, 84.50)	55.20 (41.56, 69.50)	< 0.001
MCP-1, pg/mL	84.65 (66.67, 105.80)	100.80 (81.60, 113.38)	77.14 (59.90, 95.48)	< 0.001
The results are presented as the mean ± SD, median with interquartile range, or frequencies and percentages. LVR, left ventricular remodeling; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TCHO, total cholesterol; TG, triglyceride; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; FPG, fasting plasma glucose; SD, standard deviation; CV, coefficient of variance; NDR, nocturnal dipping rate; IVST, interventricular septal thickness; LVPW, left ventricular posterior wall thickness; LVDD, left ventricular end-diastolic internal dimension; LVM, left ventricular mass; LVMI, left ventricular mass index; LVH, left ventricular hypertrophy; hsCRP, hypersensitive C-reactive protein; TNF-α, tumor necrosis factor α; MCP-1, monocyte chemotactic protein-1.				

Compared to the non-LVH group, the average of 24-hour SBP, daytime SBP, nighttime SBP, and nighttime DBP, as well as 24-hour SBP SD and CV were significantly higher in the LVH group (all *p* < 0.05). Conversely, SBP and DBP NDR in the LVH group were markedly lower than those in the non-LVH group (all *p* < 0.001). The prevalence of reverse dippers was significantly higher, whereas the prevalence of dippers and extreme dippers were lower, in the LVH group than those in the non-LVH group (*p* < 0.001). The differences in the prevalence of non-dippers were not significant between the LVH and non-LVH groups. For inflammation profiles, the plasma levels of hsCRP, TNF-α, and MCP-1 were obviously high in the LVH group compared to the non-LVH group (all *p* < 0.001; Table 1).

Correlation between BP fluctuation and LVR

Correlation analysis revealed that IVST, LVPW, and LVMI were positively related to 24-hour SBP SD and CV, whereas negatively correlated with SBP and DBP NDR (all *p* < 0.05; **Supplementary Table 1**). These significant associations remained after adjustment for confounders, even including office SBP and DBP, histories of hypertension and antihypertensive medication use (Table 2).

Table 2
Multiple linear regression analysis results.

	IVST, mm		LVPW, mm		LVMI, g/m ²		hsCRP, mg/dL		TNF- α , pg/mL		MCP-1, pg/mL	
	β (95% CI)	<i>p</i> value	β (95% CI)	<i>p</i> value	β (95% CI)	<i>p</i> value	β (95% CI)	<i>p</i> value	β (95% CI)	<i>p</i> value	β (95% CI)	<i>p</i> value
24-hour SBP SD, mmHg	0.067 (0.053, 0.080)	< 0.001	0.055 (0.039, 0.070)	< 0.001	0.984 (0.737, 1.231)	< 0.001	0.010 (0.008, 0.012)	< 0.001	0.915 (0.734, 1.096)	< 0.001	0.908 (0.678, 1.139)	< 0.001
24-hour DBP SD, mmHg	0.039 (-0.002, 0.079)	0.060	-0.002 (-0.048, 0.043)	0.923	0.272 (-0.457, 1.000)	0.464	0.012 (0.006, 0.019)	< 0.001	1.019 (0.475, 1.563)	< 0.001	0.868 (0.194, 1.541)	0.012
24-hour SBP CV, %	0.078 (0.059, 0.096)	< 0.001	0.061 (0.040, 0.082)	< 0.001	1.119 (0.781, 1.457)	< 0.001	0.013 (0.010, 0.016)	< 0.001	1.264 (1.021, 1.507)	< 0.001	1.160 (0.849, 1.472)	< 0.001
24-hour DBP CV, %	0.028 (-0.001, 0.057)	0.062	-0.005 (-0.038, 0.028)	0.781	0.194 (-0.333, 0.720)	0.470	0.009 (0.004, 0.013)	< 0.001	0.775 (0.382, 1.167)	< 0.001	0.650 (0.164, 1.137)	0.009
SBP NDR, %	-0.050 (-0.064, -0.037)	< 0.001	-0.047 (-0.063, -0.032)	< 0.001	-0.840 (-1.084, -0.596)	< 0.001	-0.004 (-0.422, -0.039)	< 0.001	-0.231 (-0.422, -0.039)	0.018	-0.519 (-0.752, -0.287)	< 0.001
DBP NDR, %	-0.084 (-0.115, -0.053)	< 0.001	-0.074 (-0.110, -0.039)	< 0.001	-1.079 (-1.643, -0.515)	< 0.001	-0.011 (-0.016, -0.006)	< 0.001	-0.391 (-0.822, -0.040)	0.075	-1.087 (-1.611, -0.564)	< 0.001
HsCRP, mg/dL	2.116 (1.629, 2.603)	< 0.001	1.909 (1.346, 2.471)	< 0.001	43.254 (34.639, 51.869)	< 0.001	–	–	–	–	–	–
TNF- α , pg/mL	0.024 (0.018, 0.030)	< 0.001	0.021 (0.015, 0.028)	< 0.001	0.537 (0.434, 0.641)	< 0.001	–	–	–	–	–	–
MCP-1, pg/mL	0.019 (0.015, 0.024)	< 0.001	0.016 (0.010, 0.021)	< 0.001	0.348 (0.261, 0.435)	< 0.001	–	–	–	–	–	–
Models were adjustment for confounders including age, sex, smoking, alcohol consumption, regular exercise, histories of hypertension, dyslipidemia, and diabetes, and corresponding medication use, BMI, office SBP and DBP, TCHO, TG, HDL-c, LDL-c, and FPG.												
SBP, systolic blood pressure; DBP, diastolic blood pressure; SD, standard deviation; CV, coefficient of variance; NDR, nighttime decline rate; IVST, interventricular septal thickness; LVPW, left ventricular posterior wall thickness; LVMI, left ventricular mass index; LVH, left ventricular hypertrophy; hsCRP, hypersensitive C-reactive protein; TNF- α , tumor necrosis factor α ; MCP-1, monocyte chemotactic protein-1, BMI, body mass index; TCHO, total cholesterol; TG, triglyceride; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; FPG, fasting plasma glucose.												

The RCS analytic model revealed positive linear dose-response correlations between LVH risk and 24-hour SBP SD and CV (all *p* for overall < 0.001, *p* for nonlinear = 0.862, and 0.795, respectively; Fig. 2). The dose-response correlations between LVH risk and 24-hour DBP SD and CV were not significant. For NDR, an L-shaped correlation emerged between LVH risk and SBP NDR (*p* for overall < 0.001, *p* for nonlinear = 0.042), while a negative linear correlation was identified between LVH risk and DBP NDR (*p* for overall = 0.001, *p* for nonlinear = 0.339; Fig. 2).

For circadian BP rhythm, the results of logistic regression showed that, compared to dippers, the risk of LVH was increased by 0.874-fold (OR: 1.874, 95% CI: 1.113 to 3.155; *p*_{adjusted} = 0.018) in non-dippers and by 3.965-fold (OR: 4.965, 95% CI: 2.847 to 8.659; *p*_{adjusted} < 0.001) in reverse dippers, after adjustment for confounders. The differences in the risk of LVH were not significant between extreme dippers and dippers (OR: 0.892, 95% CI: 0.406 to 1.958; *p*_{adjusted} = 0.776).

Correlation between inflammation and LVR

Correlation analysis revealed that IVST, LVPW, and LVMI were positively correlated with the plasma levels of hsCRP, TNF- α , and MCP-1 (all *p* < 0.05; **Supplementary Table 2**). After adjustment for confounders, the significant correlations still maintained which assess using a multiple linear regression analytic model (all *p*_{adjusted} < 0.05; Table 2). The RCS analytic model emerged linear and positive dose-response correlations between the risk of LVH and the plasma levels of hsCRP, TNF- α , and MCP-1 (*p* for nonlinear = 0.184, 0.199, and 0.355, respectively; Fig. 3).

Correlation between BP fluctuation and inflammation

Correlation analysis demonstrated that SD and CV of 24-hour SBP and DBP were positively related to, while SBP and DBP NDR were negatively related to the plasma levels of hsCRP, TNF- α , and MCP-1 (all $p < 0.05$; **Supplementary Table 3**). The correlations remained after adjustment for confounders (all $p_{adjusted} < 0.05$; Table 2).

Mediating effect of chronic inflammation on the correlation between BP fluctuation LVR

The results of mediation analyses showed that hsCRP, TNF- α , and MCP-1 collectively exerted a mediating effect on the correlation between BP fluctuation and LVR (Figs. 4 and 5). HsCRP, TNF- α , and MCP-1 contributed 22.4%, 19.4%, and 17.9% of the total effect on the correlation between 24-hour SBP SD and IVST, respectively. The corresponding contributions were 25.5%, 23.6%, and 18.2% for the correlation between 24-hour SBP SD and LVPW, and 35.9%, 40.7%, and 24.4% for the correlation between 24-hour SBP SD and LVMI (Fig. 4). Similar significant contributions from hsCRP, TNF- α , and MCP-1 were found for the correlations between 24-hour SBP CV and IVST, LVPW, and LVMI (Fig. 4).

For the correlation between SBP NDR and IVST, the contributions of hsCRP, TNF- α , and MCP-1 to the total effect were 15.7%, 9.8%, and 17.6%, respectively; the corresponding contributions were 14.9%, 10.6%, and 14.9% for the correlation between SBP NDR and LVPW, and 20.2%, 13.8%, and 14.9% for the correlation between SBP NDR and LVMI (Fig. 5). HsCRP, TNF- α , and MCP-1 contributed 25.0%, 10.7%, and 22.6% of the total effect for the correlation between DBP NDR and IVST, respectively; 25.7%, 10.8%, and 20.3% for the correlation between DBP NDR and LVPW, and 41.0%, 19.0%, and 33.1% for the correlation between DBP NDR and LVMI (Fig. 5).

Sensitivity analysis

Besides adjustment for confounders in regression analytical models, the VIF values of all covariates were assessed to limit the bias induced by multicollinearity (Table 3). These VIF values were all lower than 5, demonstrating that all covariates exhibited low multicollinearity and high independence. All covariates can be accounted for as independent confounders in the regression analytical model.

Table 3
VIF values of covariates.

Age, years	VIF value
	1.133
Female, n (%)	1.354
Hypertension, n (%)	3.445
Dyslipidemia, n (%)	1.884
Diabetes, n (%)	1.159
Smoking, n (%)	1.711
Alcohol consumption, n (%)	1.497
Regular exercise, n (%)	1.067
Anti-hypertensive medication use, n (%)	2.870
Anti-dyslipidemic medication use, n (%)	2.541
BMI, kg/m ²	1.149
Office SBP, mmHg	2.395
Office DBP, mmHg	1.364
TG, mmol/L	1.158
HDL-c, mmol/L	1.200
LDL-c, mmol/L	1.276
FPG, mmol/L	1.191
The results are presented as the mean $\pm$ SD, median with interquartile range, or frequencies and percentages. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triglyceride; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; FPG, fasting plasma glucose.	

Discussion

The main findings of this study were: Independent of 24-hour average SBP levels, 24-hour SBP SD and CV were significantly and positively related to IVST, LVPW, and LVMI in community-dwelling older adults, while SBP and DBP NDR were negatively associated with these parameters. LVH risk showed positive linear dose-response correlations with 24-hour SBP SD and CV, an L-shaped correlation with SBP NDR, and a negative linear correlation with

DBP NDR. Compared to those with a dipper BP rhythm, participants with a reverse dipper BP rhythm had a significantly increased LVH risk, followed by those with a non-dipper rhythm. Moreover, this study further demonstrated that chronic low-grade inflammation, comprehensively assessed using hsCRP, TNF- α , and MCP-1, exerted a crucial mediating effect on the correlation between BP fluctuation and LVR risk.

Tadic M, et al. [22] demonstrated that BPV, whether short-term or long-term, when assessed via 24-hour ABPM, was closely associated with early ventricular remodeling, which was detected by global and free-wall right ventricular longitudinal strain in pregnant women. In this study, participants were mainly older adults aged 60 years and above enrolled from the community. Our data showed that elevated SD and CV of 24-hour SBP were significantly associated with increased IVST, LVPW, and LVMI independent of 24-hour average SBP. Furthermore, we found positive linear dose-response correlations between LVH risk and the SD and CV of 24-hour SBP. The findings indicate that elevated BPV is an independent risk factor of LVR. Reducing BPV may be as important as lowering blood pressure levels for hypertension management in older adult.

Non-dippers and reverse dippers are characterized by low BP NDR, and manifest higher nighttime BP levels that even exceed those during the daytime. Elevated nighttime BP imposes an additional excessive stress load on the heart. Long-term sustained excessive stress load is a vital contributor to myocardial cell hyperplasia and hypertrophy, ultimately leading to LVH [23, 24]. In this study, we found that SBP NDR was negatively associated with IVST, LVPW, and LVMI in older adults. For LVH risk, an L-shaped correlation with SBP NDR. This finding indicates that LVH risk decreases significantly as SBP NDR increases. However, when NDR exceeds a certain threshold, LVH risk does not decrease significantly with increasing NDR. The results of logistic analysis also yielded similar findings in this study. Compared to the dipper rhythm, LVH risk was significantly increased in the non-dipper and reverse dipper rhythms, but no significant change was observed in the extreme dipper rhythm. Additionally, DBP NDR exhibited a significant negative correlation with LVR. This finding suggests that not only SBP NDR but also DBP NDR should be seriously considered for the health management of older adults.

Chronic low-grade inflammation may play an important underlying role in myocardial cell proliferation and hypertrophy induced by excessive BPV and abnormal circadian BP rhythm. Chronic inflammation has been recognized as a contributor to cardiac remodeling, and inflammatory markers are elevated in individuals with higher BPV and abnormal circadian BP rhythms, including non-dippers and reverse dippers [15–17, 25, 26]. In this study, hsCRP, TNF- α , and MCP-1 collectively exerted a significant indirect effect on the associations of SD and CV of 24-hour SBP, NDR of SBP and DBP with IVST, LVPW, and LVMI. The findings demonstrated that chronic low-grade inflammation plays an important mediating role in the association between abnormal BP fluctuation, such as excessive variability and lowered NDR, and LVR. These results suggest that chronic low-grade inflammation may emerge as a promising therapeutic target for inhibiting the progression of LVR induced by excessive BPV and abnormal circadian BP rhythms.

Various indices have been used to assess 24-hour BPV in previous studies [27–29], including SD, CV, weighted SD, and average real variability. Among them, weighted SD is computed as the average of daytime and nighttime BP SD weighted by the duration of each period. Its main advantage is considered to be that it is not affected by the magnitude of nocturnal BP decline. However, nocturnal BP decline is an important component of BP fluctuation, even though a normal range of decline constitutes a beneficial response of BP [28]. Average real variability is calculated as the average of absolute differences between consecutive measurements. Its advantage is that it can reflect within-subject variability, but it is more affected by low-quality data [29]. In this study, the main reason for using 24-hour SD and CV to assess BPV is that SD and CV are reliable, valid, and the most commonly used indices for all types of BPV.

The primary strength of this study was that BP fluctuations were comprehensively assessed in terms of both BP variability and circadian rhythm. Exploring the potential mediating role of chronic low-grade inflammation in the correlation between BP fluctuation and LVR represents another strength. In addition, the recruitment of study participants from the community-dwelling population can also be counted as a strength of this study.

However, several limitations of this study should be acknowledged. Firstly, due to the cross-sectional and observational study design, this study can only elucidate the correlation between BP fluctuation and LVR, but cannot establish a causal association between them. Secondly, participants were primarily recruited from Han nationality in the Shandong area of China. Thus, our findings may not be generalized or applicable to other ethnic groups among older adults [30]. Thirdly, although exercise has been accounted for as a confounding factor in statistical adjustment, environmental factors, lifestyle, and psychological states that influence BP fluctuation or LVR were not fully considered in this study. This may have introduced bias into the study findings [27, 31]. Additionally, other common comorbidities such as chronic obstructive sleep apnea and anemia, which could influence blood pressure fluctuations or ventricular remodeling, were not taken into account in this study [32, 33].

In conclusion, excessive systolic BPV and abnormal circadian BP rhythms, especially reverse dipper rhythm, are pivotal risk factors for LVR in older adults, independent of 24-hour average BP levels. Chronic low-grade inflammation exerts a significant mediating effect on these correlations. However, large-sample prospective cohort studies and clinical trials involving multiethnic groups as well as young and middle-aged individuals are needed in the future.

Abbreviations

LVR
left ventricular remodeling
BP
blood pressure

BPV
blood pressure variability
ABPM
ambulatory blood pressure monitoring
hsCRP
hypersensitive C-reactive protein
TNF- α
tumor necrosis factor α
MCP-1
monocyte chemotactic protein-1
STROBE
Strengthening the Reporting of Observational Studies in Epidemiology
SD
standard deviation
CV
coefficient of variance
NDR
nocturnal dipping rate
SBP
systolic blood pressure
DBP
diastolic blood pressure
IVST
interventricular septal thickness
LVPW
left ventricular posterior wall thickness
LVDD
left ventricular end-diastolic internal dimension
LVMI
left ventricular mass index
LVH
left ventricular hypertrophy
BMI
body mass index
TCHO
total cholesterol
TG
triglyceride
HDL-c
high-density lipoprotein cholesterol
LDL-c
low-density lipoprotein cholesterol
FPG
fasting plasma glucose
RCS
restricted cubic spline
IQR
interquartile range
OR
odds ratio
CI
confidence interval
VIF
variance inflation factor.

Summary

What is known about this topic?

- Due to changes in physiological functions, BP fluctuations in the elderly are alerted, leading to excessive variability and abnormal circadian rhythms.
- Elevated average BP is one of the important risk factors of LVR.
- Chronic low-grade inflammation is closely associated with LVR.

What is new?

- Beyond elevated average levels, abnormal fluctuation of BP including excessive variability and abnormal circadian rhythm plays a crucial role in LVR.
- Not only SBP NDR but also DBP NDR is closely and negatively related to LVR.
- Chronic low-grade inflammation mediates the process by which abnormal BP fluctuation inducing LVR.

Declarations

Data Availability Statement

Data is available from the corresponding author upon reasonable request.

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Author contributions

Chunju Zhang and Zhendong Liu designed the study. Xiaojing Zhang, Jia Liu, Peilin Li, Siyi Dun, and Zhendong Liu performed statistical analysis. Xiaojing Zhang drafted the manuscript. All the authors collected the data and reviewed and revised the manuscript for intellectual content and approved the final version.

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Ethical Approval

This study was conducted in compliance with the Declaration of Helsinki and was approved by the Research Ethics Committee of the Institute of Basic Medicine, Shandong Academy of Medical Sciences, Jinan, China. Written informed consent was obtained from each participant.

Competing interests

No potential conflict of interest was reported by the author(s).

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Figures

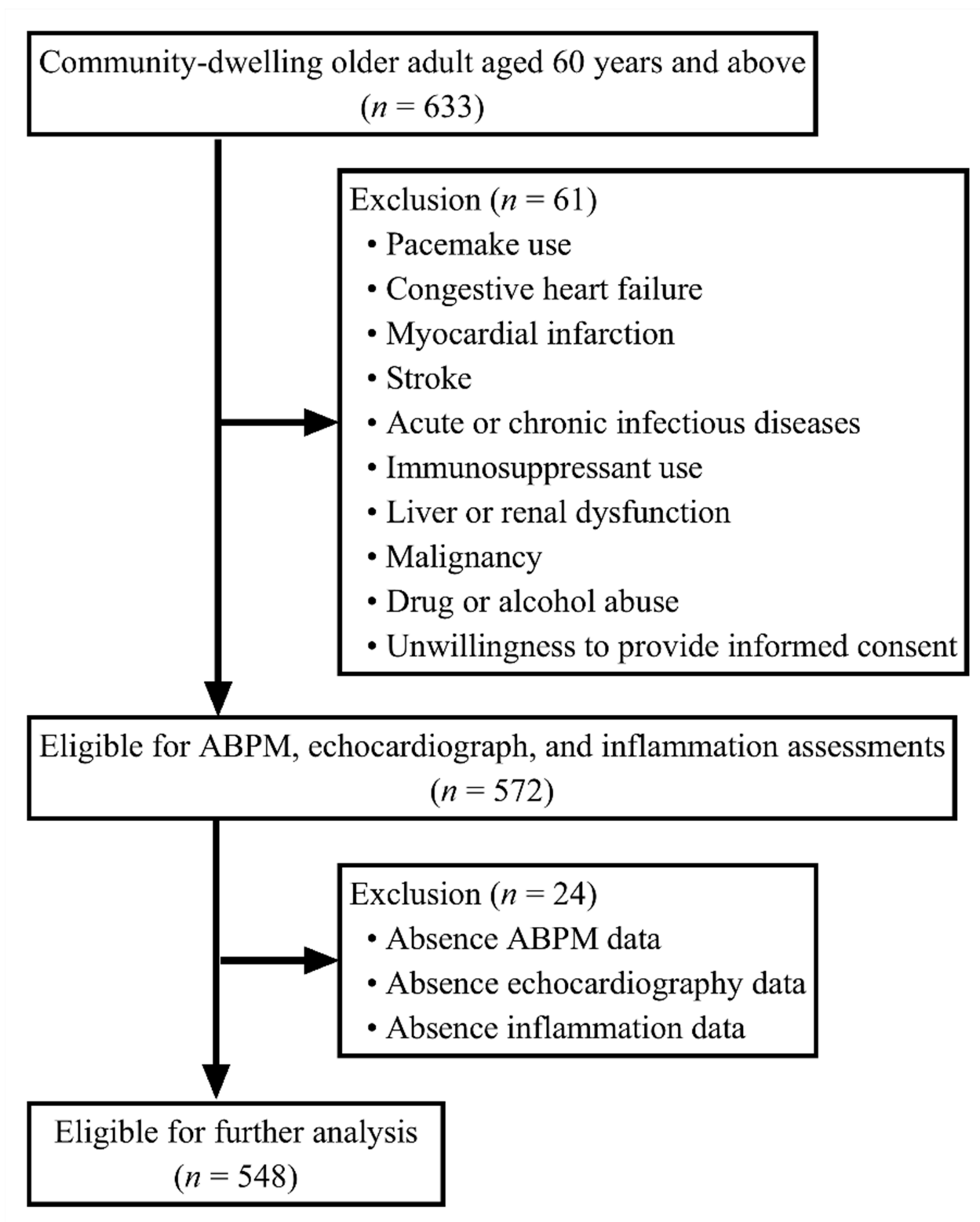


Figure 1

Study flowchart. ABPM, ambulatory blood pressure monitoring.

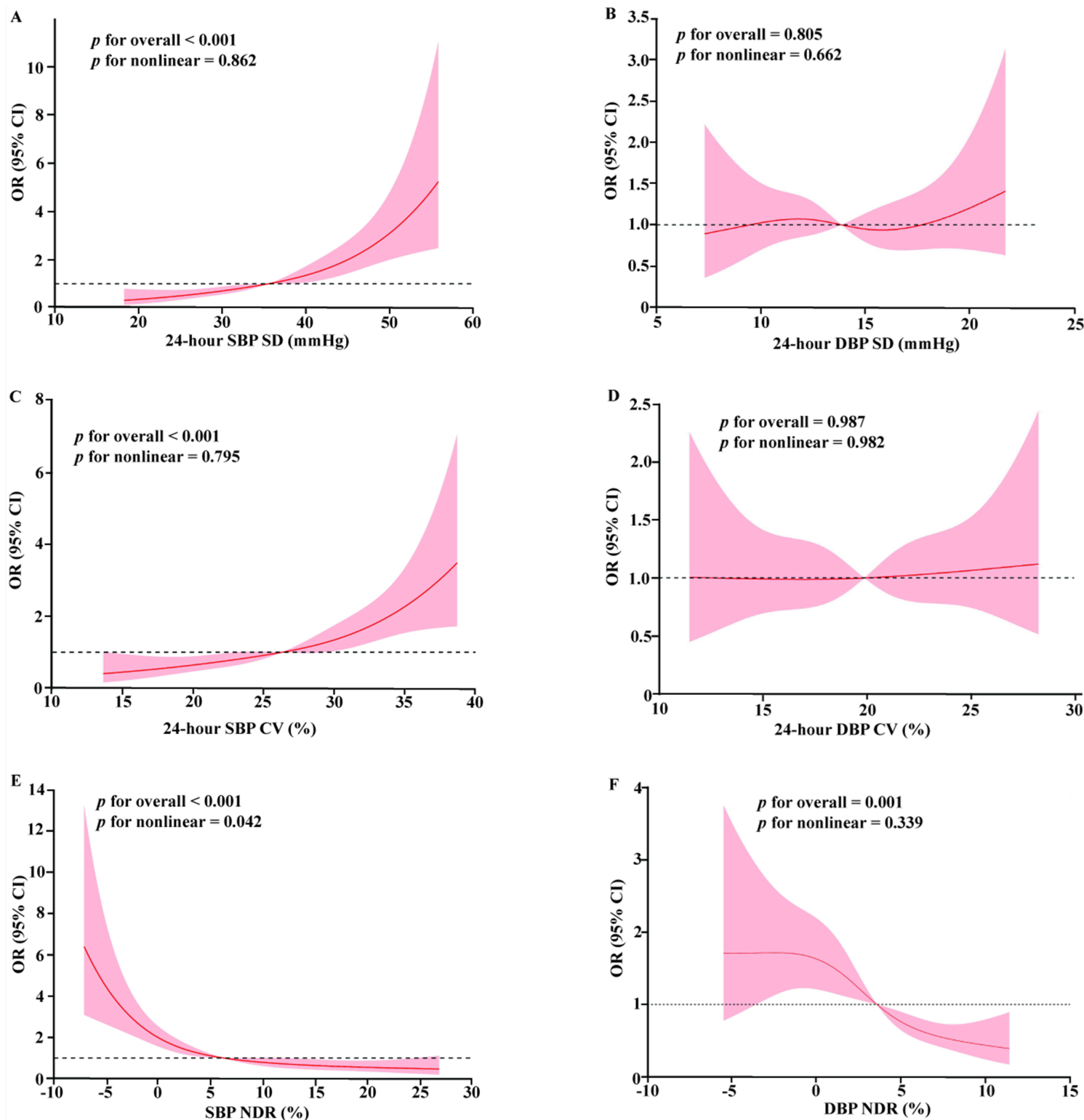


Figure 2

Dose-response correlation between BP fluctuation and LVH risk. (A) Dose-response correlation between 24-hour SBP SD and LVH risk. (B) Dose-response correlation between 24-hour DBP SD and LVH risk. (C) Dose-response correlation between 24-hour SBP CV and LVH risk. (D) Dose-response correlation between 24-hour DBP CV and LVH risk. (E) Dose-response correlation between SBP NDR and LVH risk. (F) Dose-response correlation between DBP NDR and LVH risk. SBP, systolic blood pressure; DBP, diastolic blood pressure; SD, standard deviation; CV, coefficient of variance; NDR, nocturnal dipping rate; OR, odds ratio.

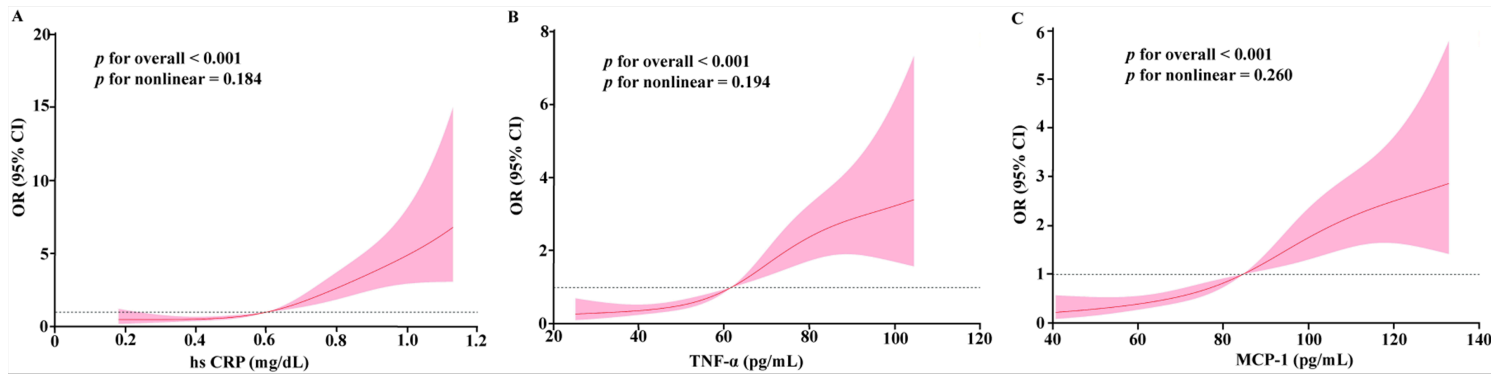


Figure 3

Dose-response correlation between inflammation and LVH risk. (A) Dose-response correlation between hsCRP and LVH risk. (B) Dose-response correlation between TNF-α and LVH risk. (C) Dose-response correlation between MCP-1 and LVH risk. OR, odds ratio; hsCRP, hypersensitive C-reactive protein; TNF-α, tumor necrosis factor α; MCP-1, monocyte chemotactic protein-1.

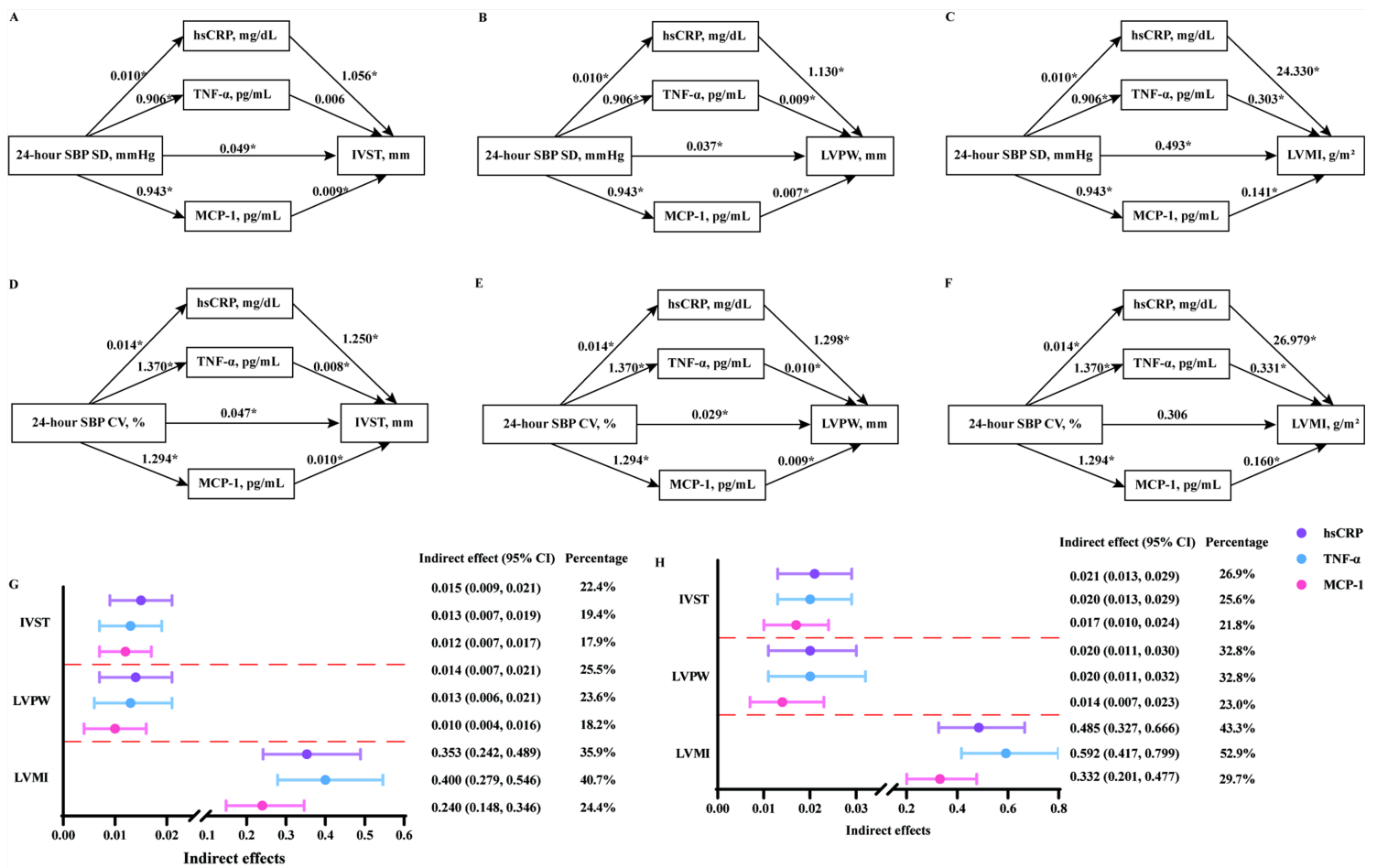


Figure 4

Mediating effects chronic low-grade inflammation on the correlation between BPV and LVR. (A-C) Collective mediating effect of hsCRP, TNF-α, and MCP-1 on the correlation between 24-hour SBP SD and IVST (A), LVPW (B), and LVMI (C). (D-F) Collective mediating effect of hsCRP, TNF-α, and MCP-1 on the correlation between 24-hour SBP CV and IVST (D), LVPW (E), and LVMI (F). (G) The indirect effects and percentage of hsCRP, TNF-α, and MCP-1 on the correlations between 24-hour SBP SD and IVST, LVPW, and LVMI. (H) The indirect effects of hsCRP, TNF-α, and MCP-1 on the correlations between 24-hour SBP CV and IVST, LVPW, and LVMI. SBP, systolic blood pressure; SD, standard deviation; CV, coefficient of variance; IVST, interventricular septal thickness; LVPW, left ventricular posterior wall thickness; LVMI, left ventricular mass index; hsCRP, hypersensitive C-reactive protein; TNF-α, tumor necrosis factor α; MCP-1, monocyte chemotactic protein-1. *, p < 0.05.

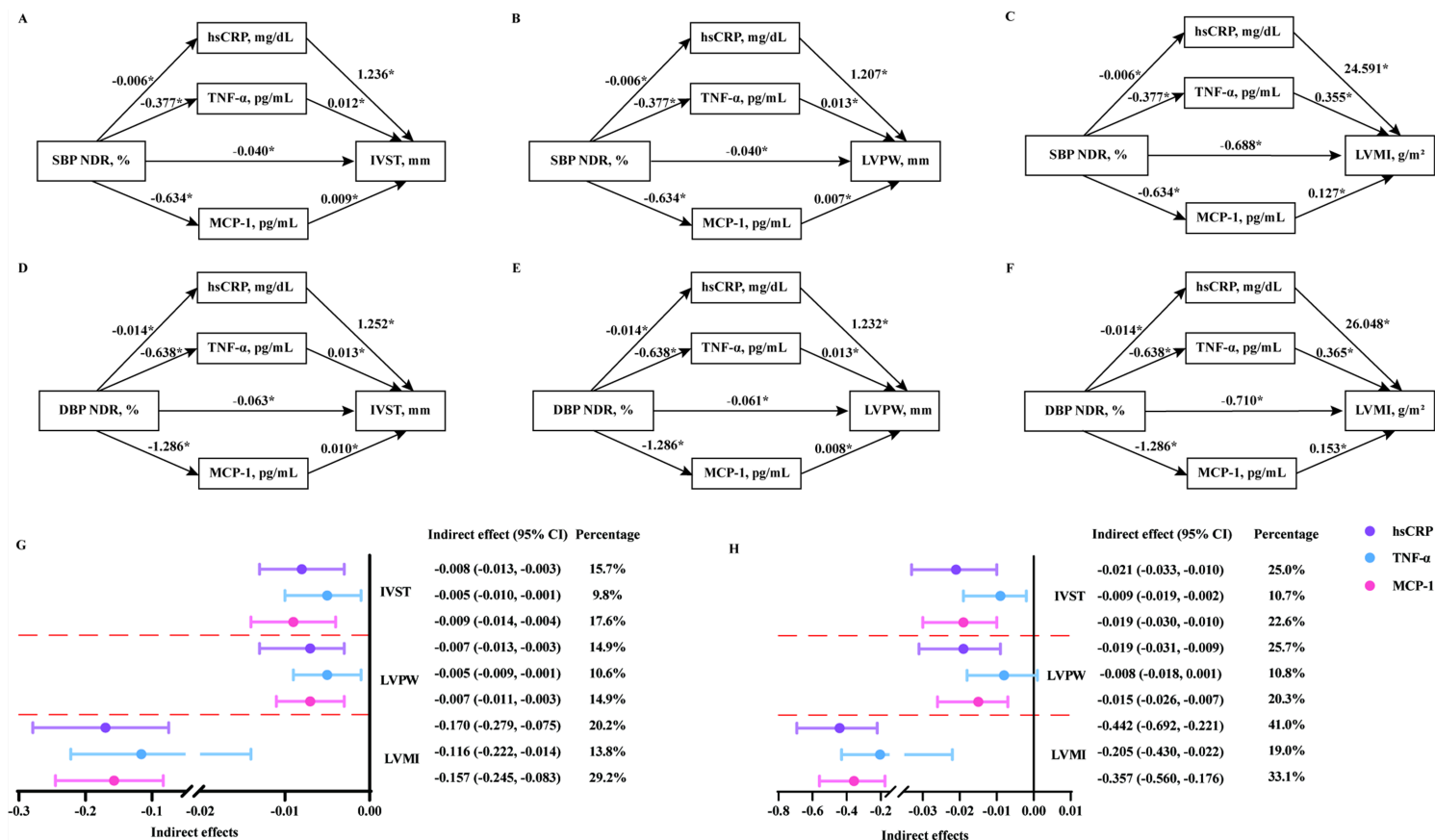


Figure 5

Mediating effect of chronic low-grade inflammation on the correlation between BP NDR and LVR. (A-C) Collective mediating effect of hsCRP, TNF-α, and MCP-1 on the correlation between SBP NDR and IVST (A), LVPW (B), and LVMI (C). (D-F) Collective mediating effect of hsCRP, TNF-α, and MCP-1 on the correlation between DBP NDR and IVST (D), LVPW (E), and LVMI (F). (G) The indirect effects and percentage of hsCRP, TNF-α, and MCP-1 on the correlations between SBP NDR and IVST, LVPW, and LVMI. (H) The indirect effects of hsCRP, TNF-α, and MCP-1 on the correlations between DBP NDR and IVST, LVPW, and LVMI. SBP, systolic blood pressure; NDR, nighttime decline rate; IVST, interventricular septal thickness; LVPW, left ventricular posterior wall thickness; LVMI, left ventricular mass index; hsCRP, hypersensitive C-reactive protein; TNF-α, tumor necrosis factor α; MCP-1, monocyte chemotactic protein-1. *, $p < 0.05$.

Supplementary Files

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