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Nonlinear association between LDL/HDL ratio and hypertension: a cross-sectional analysis based on Chinese adults

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How to cite this article: Lai W, Chen X, Wang L, Li X, Liu M, Wu L, Zhou B. Nonlinear association between LDL/HDL ratio and hypertension: a cross-sectional analysis based on Chinese adults. *J Cardiovasc Aging*. 2025;5:12. <https://dx.doi.org/10.20517/jca.2024.35>

Received: 2 Dec 2024 **First Decision:** 25 Apr 2025 **Revised:** 12 May 2025 **Accepted:** 5 Jun 2025 **Published:** 13 Jun 2025

Academic Editor: Lemin Zheng **Copy Editor:** Fangling Lan **Production Editor:** Fangling Lan

Abstract

Introduction: The link between low-density lipoprotein/high-density lipoprotein (LDL/HDL ratio, LHR) and the prevalence of hypertension in large populations, especially among individuals aged 20 and older, has not been extensively explored. This research aims to examine whether LHR is associated with hypertension prevalence.

Methods: This retrospective cohort analysis drew on cross-sectional data from a health check-up database located in China. Hypertension is classified as a systolic blood pressure (SBP) of ≥ 140 mmHg or a diastolic blood pressure (DBP) of ≥ 90 mmHg. Multivariable logistic regression was used to examine the association between LHR and hypertension prevalence. Furthermore, a subgroup analysis was performed to assess how this association varied across different demographic categories.



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Results: The cross-sectional evaluation encompassed 113,912 participants. After adjusting for confounding variables, every unit rise in LHR correlated with an 11% increase in the prevalence of hypertension (Odds Ratio [OR] = 1.11, 95% Confidence Interval [CI]: 1.06, 1.16). Our findings indicated a nonlinear relationship between LHR and hypertension prevalence, with an inflection point at 2.28. Below this level, each unit increase in LHR was linked to a 168% heightened risk of hypertension, while above this point, the correlation became insignificant. The subgroup analysis indicated that females, older adults, non-smokers, and individuals with lower body mass index (BMI) exhibited a particularly high prevalence of hypertension associated with higher LHR levels.

Conclusion: The findings suggest that a higher LHR serves as a notable predictor of hypertension and elevated blood pressure among Chinese adults aged 20 and older. The identified nonlinear relationship and threshold effect highlight the importance of LHR in hypertension screening and management.

Keywords: LDL/HDL ratio, hypertension, prevalence

INTRODUCTION

Hypertension serves as a primary contributor to fatalities and disability-adjusted life years globally, emerging as one of the most prevalent and swiftly proliferating cardiovascular diseases (CVD) in recent times^[1]. Predictions indicate that by 2025, there will be a notable increase in the prevalence of hypertension worldwide, particularly among the adult population, where, despite advancements in awareness, treatment, and management, the overall frequency of hypertension is still on the rise^[2]. This growing prevalence correlates with a rising incidence and mortality associated with CVD^[3,4]. Among the significant pathological mechanisms that contribute to hypertension, atherosclerosis has received considerable attention, with lipid metabolism playing a vital role in this mechanism^[5].

Within the indicators of lipid metabolism, the ratio of low-density lipoprotein cholesterol (LDL-C)/high-density lipoprotein cholesterol (HDL-C) - termed LDL/HDL ratio (LHR) - is acknowledged as a critical marker for assessing cardiovascular health^[6]. Research indicates that LHR not only adeptly predicts cardiovascular events, but its predictive efficacy even exceeds that of measuring HDL-C or LDL-C levels in isolation^[7]. Additionally, LHR is intricately linked with all-cause mortality among hypertensive individuals, a relationship that is particularly acute in older populations^[8]. Nevertheless, despite these insights, the significance of LHR in diverse populations across different age categories has not been comprehensively explored^[9]. This dataset enables a robust evaluation of the association between LHR and hypertension^[10].

This study aims to examine the relationship between LHR and the prevalence of hypertension in adults aged 20 years and older. To accomplish this, we carried out a cross-sectional analysis utilizing data from the Chinese Rich Healthcare Group, a health check-up study that is nationally representative in China. This dataset enables a robust evaluation of the association between LHR and hypertension.

METHODS

Study design

This research employed a computerized health check-up database in China created by Rich Healthcare, encompassing 32 regions within 11 provinces, which exemplifies a broad geographic distribution across the nation. The database comprises demographic details, health status, and information related to blood tests and functional assessments. Baseline data were sourced from the health check-up records of 685,277 individuals at Rich Healthcare centers from 2010 to 2016. Initially, 211,833 participants ≥ 20 years old who had at least two visits during this timeframe were included. The dataset is accessible on the official website, www.datadryad.org, where it can be downloaded ([doi:10.5061/dryad.ft8750v](https://doi.org/10.5061/dryad.ft8750v)).

In our cross-sectional research, we set the subsequent criteria for inclusion: individuals who are at least 20 years old; full diagnostic information on hypertension, which encompasses LDL-C, HDL-C, systolic blood pressure (SBP), diastolic blood pressure (DBP), and comprehensive sociodemographic data. Despite the Rich Healthcare database including individuals with a minimum of two visits to maintain longitudinal integrity, only the baseline data from the first visit were utilized in this cross-sectional analysis. The exclusion criteria included: incomplete hypertension diagnostic data, HDL-C, or LDL-C; and outliers for SBP, DBP, LDL-C, or HDL-C [values above the mean plus three standard deviations (SD) or below the mean minus three SD]. Following thorough screening, a total of 113,912 participants were deemed eligible for the cross-sectional analysis, as illustrated in [Figure 1](#).

Data collection

Data were collected by professionals after proper training utilizing a structured questionnaire. Participants needed to fill out a comprehensive questionnaire aimed at gathering sociodemographic data. Health-related behaviors, including smoking and alcohol drinking, along with medical history and family backgrounds regarding chronic illnesses, were also documented. Qualified professionals conducted physical assessments, which included height, weight, body mass index (BMI), and blood pressure. Blood pressure readings were taken using a standard mercury sphygmomanometer. Blood samples were collected from the vein during the early morning hours when fasting to evaluate levels of total cholesterol (TC), LDL-C, HDL-C, triglycerides (TG), fasting blood glucose (FBG), serum creatinine (SCr), and blood urea nitrogen (BUN), aspartate aminotransferase (AST), and alanine aminotransferase (ALT). The covariates considered in this study included age, smoking habits, alcohol consumption, FBG, BUN, SCr, and levels of ALT and AST.

Measurement of LHR

The LHR was determined^[11] as LDL-C/HDL-C, with both values expressed in mmol/L. In the following analysis, LHR was evaluated both as a continuous variable and as a categorical variable, divided into three groups to strengthen the analytical framework: Q1 ($\text{LHR} \leq 1.76$), Q2 ($1.76 < \text{LHR} \leq 2.24$), and Q3 ($\text{LHR} > 2.24$).

Definition of hypertension

Hypertension was defined as meeting at least one of the subsequent criteria: a SBP ≥ 140 mmHg or a DBP ≥ 90 mmHg^[12].

Statistical analysis

Continuous data were represented as means \pm SD, while categorical data were displayed as counts and percentages. To assess differences among the LHR groups, appropriate statistical tests were applied, including the Kruskal-Wallis H test, chi-square test, and one-way ANOVA. Multiple logistic regression was used to examine the association between LHR and hypertension. Three analytical models were constructed (Model 1 was unadjusted; Model 2 adjusted for covariates such as smoking, age, and alcohol consumption; Model 3 further adjusted for biochemical markers including FBG, SCr, BUN, ALT, and AST, in addition to the variables in Model 2). Analyses of subgroups were performed according to gender, age, alcohol drinking, smoking, and BMI, with interactions evaluated through multivariate logistic regression models. To explore potential nonlinear relationships, a generalized additive model (GAM) was utilized, and segmented regression was applied to identify potential threshold effects. $P < 0.05$ was considered statistically significant. Missing values for covariates were addressed through multiple imputations utilizing the Fully Conditional Specification (FCS) technique to minimize bias and ensure robust statistical power. Sensitivity analyses indicated that imputed results remained consistent. Statistical analyses were carried out utilizing R software (version 4.3.1) and Empower (version 6.0).

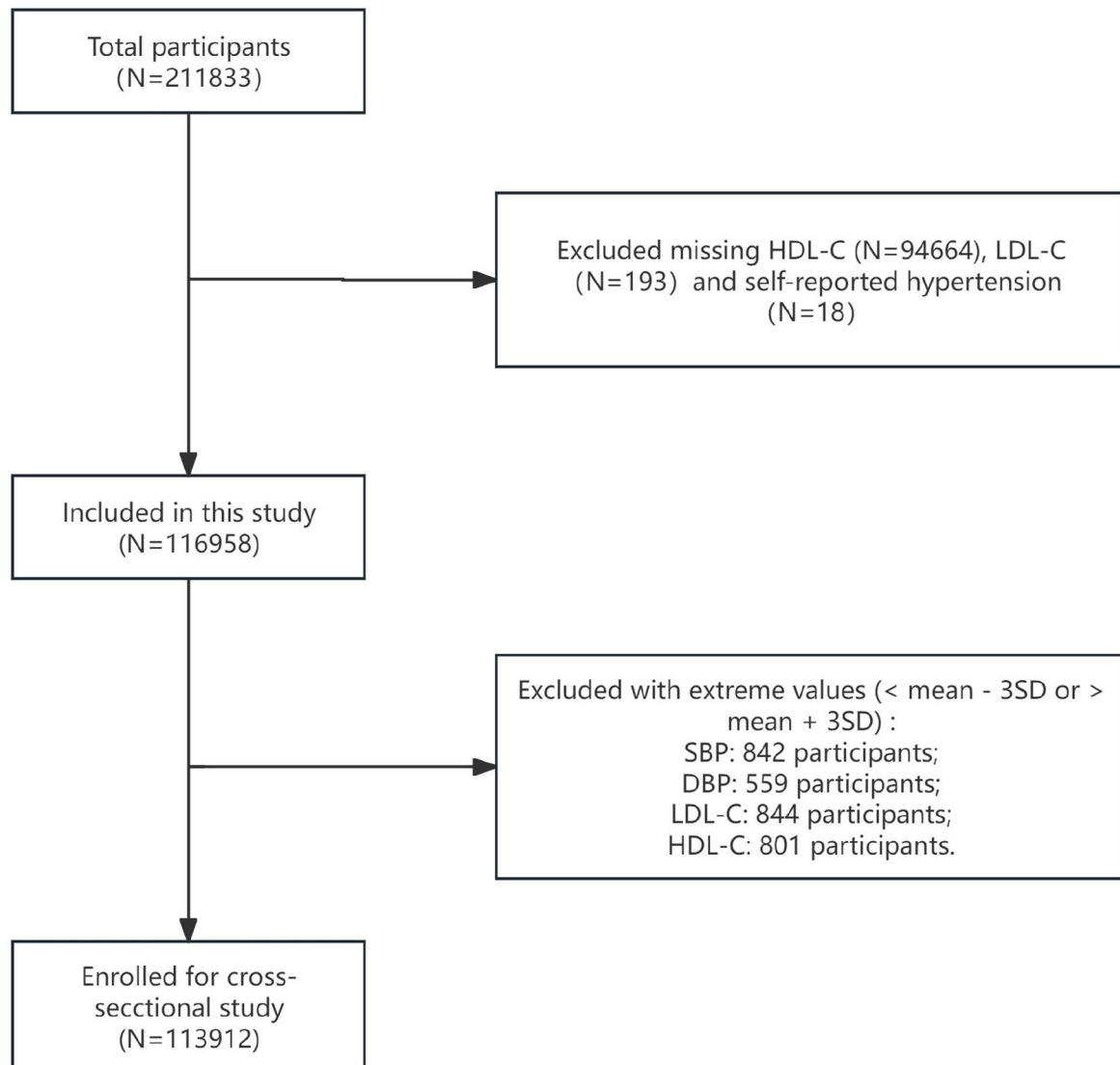


Figure 1. Flowchart of participant selection.

RESULTS

Characteristics of the participants

A total of 211,833 individuals were initially enrolled in the Rich study. After removing cases with missing and abnormal data, 113,912 participants were included in the cross-sectional analysis [Figure 1]. Among these, 15,858 individuals satisfied the diagnostic criteria for hypertension, representing roughly 13.9% of the total study cohort. Participants were categorized into three groups based on their LHR levels: Q1 ($LHR \leq 1.76$, $N = 37,971$), Q2 ($LHR 1.76-2.24$, $N = 37,963$), and Q3 ($LHR > 2.24$, $N = 37,978$). Those in the Q3 group were generally older, exhibited a higher male proportion, and had significantly elevated SBP and DBP readings. Furthermore, the Q3 group demonstrated increased levels of TC and TG. The rates of smoking and alcohol drinking were also higher in the Q3 group. Additionally, significant variations in other variables such as BMI, BUN, and serum creatinine were observed across the different groups ($P < 0.001$) [Table 1].

Table 1. Baseline characteristics of participants by LHR quartiles in the cross-sectional study

Variable	Total	Q1 ≤ 1.76	Q2 1.76-2.24	Q3 > 2.24	P value
Participants, sample size (N)	113,912	37,971	37,963	37,978	NA
Age, y	43.82 ± 12.52	40.74 ± 11.89	43.84 ± 12.67	46.87 ± 13.00	< 0.001
Gender (%)					< 0.001
male	61,457 (53.95)	14,898 (39.24)	20,626 (54.33)	25,933 (68.28)	
female	52,455 (46.05)	23,073 (60.76)	17,337 (45.67)	12,045 (31.72)	
SBP, mmHg	118.79 ± 15.47	115.15 ± 15.11	119.20 ± 15.75	122.02 ± 15.54	< 0.001
DBP, mmHg	74.07 ± 10.24	71.57 ± 10.03	74.25 ± 10.40	76.39 ± 10.29	< 0.001
Drinking (%)					< 0.001
current	851 (2.64)	252 (2.43)	262 (2.60)	337 (2.90)	
ever	5,466 (17.04)	1,446 (13.93)	1,946 (19.32)	2,074 (17.87)	
never	25,738 (80.31)	8,681 (83.64)	7,865 (78.08)	9,192 (79.22)	
Smoking (%)					< 0.001
current	6,531 (20.06)	1,244 (11.99)	2,008 (19.93)	3,279 (28.26)	
ever	1,311 (4.06)	304 (2.93)	437 (4.34)	570 (4.91)	
never	24,213 (75.88)	8,831 (85.09)	7,628 (75.73)	7,754 (66.83)	
Height, cm	166.35 ± 8.21	164.89 ± 7.95	166.29 ± 8.39	167.86 ± 8.30	< 0.001
Weight, kg	64.85 ± 11.42	60.00 ± 10.73	65.05 ± 11.64	69.49 ± 11.88	< 0.001
BMI, kg/m ²	23.32 ± 3.10	21.98 ± 3.01	23.42 ± 3.17	24.56 ± 3.13	< 0.001
FBG, mmol/L	4.94 ± 0.60	4.84 ± 0.56	4.97 ± 0.60	5.01 ± 0.64	< 0.001
TC, mmol/L	4.75 ± 0.75	4.28 ± 0.69	4.71 ± 0.75	5.27 ± 0.81	< 0.001
TG, mmol/L	1.37 ± 0.97	1.02 ± 0.95	1.33 ± 0.94	1.75 ± 1.03	< 0.001
LDL, mmol/L	2.74 ± 0.50	2.26 ± 0.44	2.73 ± 0.47	3.24 ± 0.58	< 0.001
HDL, mmol/L	1.37 ± 0.24	1.56 ± 0.26	1.38 ± 0.23	1.16 ± 0.22	< 0.001
BUN, mmol/L	4.68 ± 1.17	4.51 ± 1.15	4.72 ± 1.18	4.80 ± 1.17	< 0.001
SCr, umol/L	47.34 ± 10.66	66.12 ± 15.14	71.09 ± 15.67	48.0 ± 1.17	< 0.001
ALT, U/L	23.74 ± 21.24	19.35 ± 18.90	23.24 ± 21.70	28.63 ± 23.13	< 0.001
AST, U/L	23.96 ± 11.35	22.64 ± 12.87	23.76 ± 9.88	25.47 ± 11.29	< 0.001

Annotation: Data are presented as mean ± standard deviation or number (%).

LHR and hypertension

Multivariate regression analysis revealed a significant association between LHR and hypertension. After adjusting for potential covariates, each one-unit increase in LHR was associated with an 11% higher risk of hypertension [odds ratios (ORs) = 1.11, 95% confidence intervals (CI): 1.06, 1.16, $P < 0.0001$]. Specifically, participants in the Q2 and Q3 groups had a significantly increased risk of hypertension compared to those in the Q1 group - 33% higher in Q2 (OR = 1.33, 95%CI: 1.24, 1.43, $P < 0.0001$) and 30% higher in (OR = 1.30, 95%CI: 1.21, 1.40, $P < 0.0001$) [Table 2].

Nonlinear relationship between LHR and hypertension

Using generalized additive models (GAM) alongside smooth curve fitting techniques, we evaluated the connection between LHR and the prevalence of hypertension, as illustrated in Figure 2. The findings revealed a nonlinear relationship between LHR and hypertension (P for nonlinear < 0.001). To further explore this association, we employed the inflection point model (Model II), which identified an inflection point at an LHR value of 2.28. Below this threshold, LHR was significantly associated with an increased risk of hypertension (OR = 2.68, 95%CI: 2.54, 2.84, $P < 0.0001$); however, above this threshold, the relationship was not statistically significant (OR = 1.01, 95%CI: 0.97, 1.05, $P = 0.7631$). The log-likelihood ratio test ($P < 0.001$) showed that Model II more accurately depicted the nonlinear relationship between LHR and hypertension [Table 3].

Table 2. Multivariate regression analysis of the association between LHR and hypertension prevalence

Variable	Model 1		Model 2		Model 3	
	OR (95%CI)	P value	OR (95%CI)	P value	OR (95%CI)	P value
LHR	1.46 (1.43, 1.50)	< 0.0001	1.26 (1.23, 1.29)	< 0.0001	1.11 (1.06, 1.16)	< 0.0001
Q1	Ref.		Ref.		Ref.	
Q2	1.72 (1.65, 1.80)	< 0.0001	1.47 (1.40, 1.54)	< 0.0001	1.33 (1.24, 1.43)	< 0.0001
Q3	2.25 (2.16, 2.36)	< 0.0001	1.64 (1.56, 1.71)	< 0.0001	1.30 (1.21, 1.40)	< 0.0001

Annotation: Model 1 adjusted for none; Model 2 adjusted for age, smoking status, and drinking status; Model 3 adjusted for FBG, SCr, BUN, ALT, and AST on the basis of Model 2; LDL/HDL ratio analyzed as a continuous variable and in three groups.

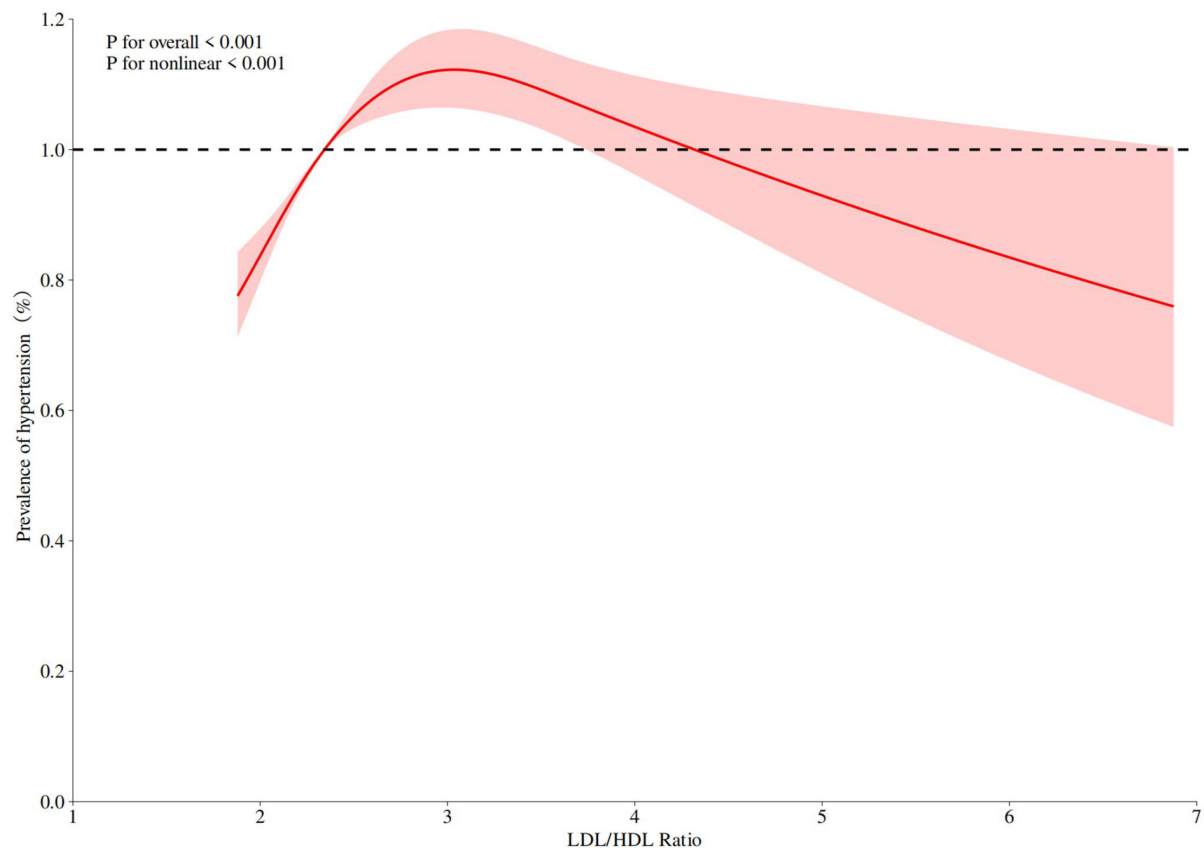


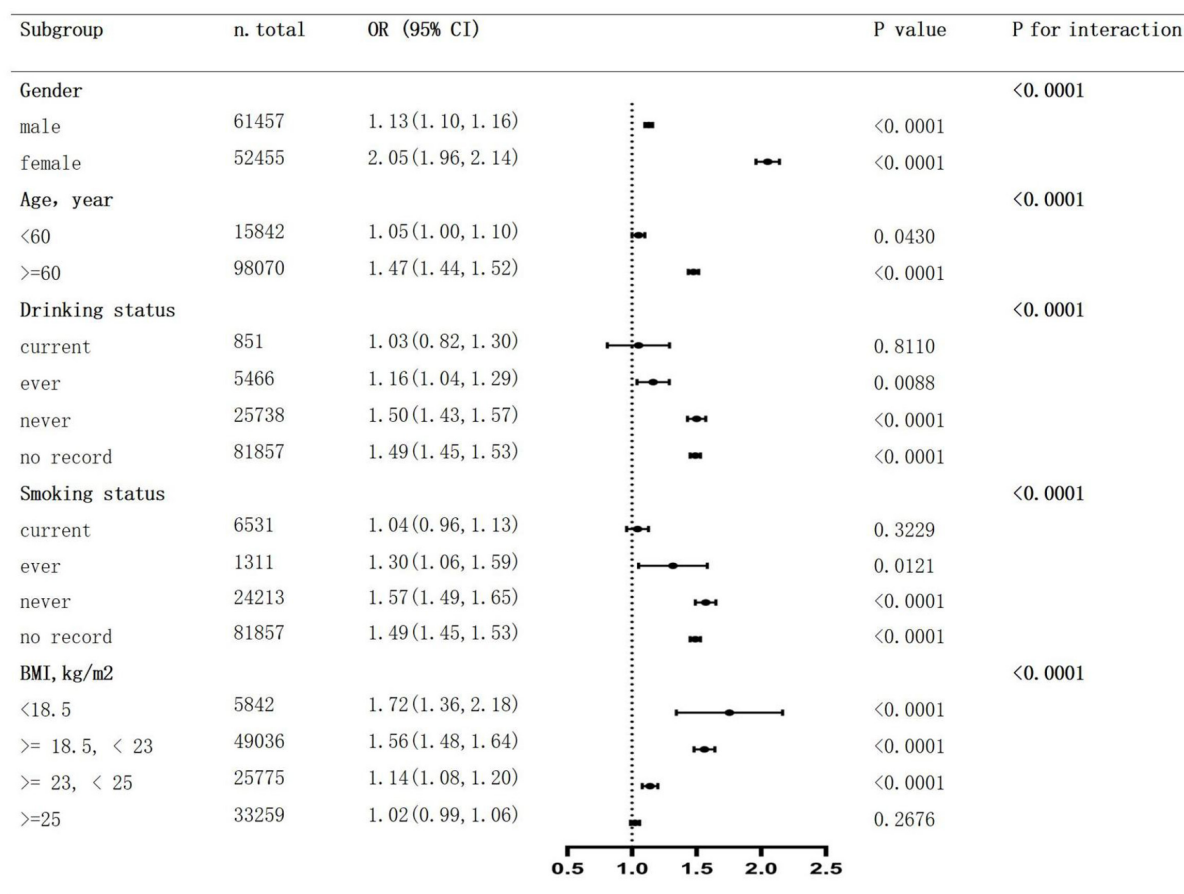
Figure 2. Smooth curve fitting was used to evaluate the linear relationship between LHR and hypertension prevalence. The red solid line represents the probability of hypertension prevalence, and the red dotted line represents the 95% confidence interval curve.

Subgroup analysis

Subgroup analyses focused on demographic and lifestyle factors [Figure 3]. Overall, the LHR-hypertension association was maintained in nearly all subgroups, reinforcing the general relevance of the main findings. Nonetheless, several significant differences in effect size were identified. The association was notably stronger among women (OR = 2.05) compared to men (OR = 1.13), and in older adults aged 60 years and above (OR = 1.47) relative to those younger than 60 (OR = 1.05). Likewise, the connection between LHR and hypertension was more significant in individuals who had never smoked or consumed alcohol, as well as in those with a lower BMI, especially among participants classified as underweight (BMI < 18.5,

Table 3. Threshold effect analysis of LHR on hypertension prevalence.

LHR	Hypertension prevalence
Model I	
Fitting by the standard linear model	1.46 (1.43, 1.50) < 0.0001
Model II	
Inflection point	2.28
< 2.28	2.68 (2.54, 2.84) < 0.0001
> 2.28	1.01 (0.97, 1.05) 0.7631
Log-likelihood ratio	< 0.001

**Figure 3.** Subgroup analysis of the association between LHR and hypertension prevalence.

OR = 1.72). The interaction effects were determined to be statistically significant (with all *P* values for interaction < 0.001), suggesting potential effect modification. These results suggest that specific populations - particularly women, older individuals, non-smokers, and those with a lower body mass - may exhibit greater vulnerability to the hypertensive impacts of increased LHR. This emphasizes the necessity for tailored cardiovascular risk evaluations and indicates that LHR could be especially useful in these higher-risk groups.

Association between LHR and blood pressure measurement

This research thoroughly investigated the connection between LHR and blood pressure measurements, emphasizing both SBP and DBP. The established criteria for hypertension diagnostics delineated the thresholds for SBP and DBP. Upon considering the pertinent covariates, a significant relationship was found, indicating that a one-unit rise in LHR was strongly associated with SBP readings of 140 mmHg or above (OR = 1.08, 95%CI: 1.03, 1.13, $P = 0.0019$) and DBP readings of 90 mmHg or more (OR = 1.23, 95%CI: 1.17, 1.29, $P < 0.0001$). These results highlight the strong association between LHR and increased blood pressure, even when multiple variables were taken into consideration. Moreover, the revised guidelines issued by the 2017 ACC/AHA and the 2022 Chinese hypertension clinical recommendations decreased the diagnostic thresholds to 130/80 mmHg, thereby accentuating cardiovascular disease risk for populations with blood pressure readings within this range. Our evaluation indicated that each unit rise in LHR was significantly related to SBP levels between 130 and 140 mmHg (OR = 1.14, 95%CI: 1.10, 1.19, $P < 0.0001$) and DBP levels between 80 and 90 mmHg (OR = 1.24, 95%CI: 1.20, 1.28, $P < 0.0001$)^[13,14]. These findings provide insight into the essential function of LHR in the regulation of blood pressure and its potential as an indicator of hypertension risk [Table 4].

DISCUSSION

This study highlights the significant association between LHR and hypertension in health check-up participants aged 20 and above. Our findings underscored the importance of lipid metabolism in the initiation of hypertension and related cardiovascular issues. A key outcome of this investigation is the persistent link observed between high LHR levels and the rising rates of hypertension. Earlier research has shown that a raised LHR serves as a reliable indicator of cardiovascular incidents and mortality^[15]. This study is among the pioneering ones to examine this link across a wide age spectrum, instead of concentrating solely on older demographics, where this connection has typically been more evident^[16]. The reliability of LHR as a marker for cardiovascular disease risk, even after accounting for various covariates, underscores its potential utility as a predictive instrument in clinical settings^[17].

The examination of the association between LHR and hypertension unveiled a multifaceted nonlinear connection. Employing GAM and smooth curve fitting methods, our analysis demonstrated that the correlation between LHR and hypertension significantly diverges from a linear trend. Notably, the threshold model (Model II) identified a crucial LHR threshold of 2.28. For values below this threshold, every unit rise in LHR correlated with a 168% heightened risk of hypertension; however, above this threshold, the association lost significance, indicating a potential saturation point or other underlying mechanisms at play. This nonlinearity highlights the necessity of considering threshold effects when assessing cardiovascular risk factors. The likelihood ratio test demonstrated that Model II more effectively captured the intricacies of the relationship between LHR and hypertension. Our results further reinforce the importance of LHR as an indicator of hypertension, especially within defined ranges, and underline the requirement for further investigations to uncover the underlying mechanisms. A potential interpretation is that slight increases in LHR could result in endothelial impairment, oxidative stress, and persistent low-grade inflammation - key mechanisms linked to the onset of hypertension. Conversely, at elevated levels of LHR, this influence might plateau due to compensatory biological responses or medical interventions in individuals at high risk, resulting in a reduced incremental risk increase. The results present significant clinical implications. The established threshold of 2.28 could be an applicable reference for healthcare providers. Those with LHR values nearing or surpassing this limit - especially within specific groups such as women, older individuals, and non-smokers - might gain from more rigorous blood pressure evaluations and proactive strategies, including changes in lifestyle or lipid management. Incorporating LHR into current cardiovascular risk assessment frameworks could improve early identification of hypertension risk, particularly in settings with limited resources or during general screenings. Moreover, these results highlight that tailored interventions might be more successful when LHR falls below the essential threshold.

Table 4. Multivariate regression analysis of LHR status and indicators of blood pressure

Variable	Model 1		Model 2		Model 3	
	OR (95%CI)	P value	OR (95%CI)	P value	OR (95%CI)	P value
Systolic pressure ≥ 140 mmHg						
LHR	1.41 (1.37, 1.45)	< 0.0001	1.19 (1.15, 1.22)	< 0.0001	1.08 (1.03, 1.13)	0.0019
Q1	Ref.		Ref.		Ref.	
Q2	1.67 (1.58, 1.76)	< 0.0001	1.38 (1.31, 1.46)	< 0.0001	1.29 (1.18, 1.40)	< 0.0001
Q3	2.16 (2.05, 2.27)	< 0.0001	1.49 (1.41, 1.57)	< 0.0001	1.27 (1.16, 1.38)	< 0.0001
Diastolic pressure ≥ 90 mmHg						
LHR	1.49 (1.45, 1.54)	< 0.0001	1.35 (1.31, 1.39)	< 0.0001	1.23 (1.17, 1.29)	< 0.0001
Q1	Ref.		Ref.		Ref.	
Q2	1.75 (1.65, 1.86)	< 0.0001	1.56 (1.47, 1.66)	< 0.0001	1.37 (1.25, 1.50)	< 0.0001
Q3	2.34 (2.21, 2.48)	< 0.0001	1.87 (1.76, 1.98)	< 0.0001	1.50 (1.36, 1.64)	< 0.0001
140 mmHg > Systolic pressure ≥ 130 mmHg						
LHR	1.36 (1.33, 1.39)	< 0.0001	1.27 (1.24, 1.30)	< 0.0001	1.14 (1.10, 1.19)	< 0.0001
Q1	Ref.		Ref.		Ref.	
Q2	1.47 (1.41, 1.53)	< 0.0001	1.37 (1.32, 1.44)	< 0.0001	1.24 (1.16, 1.33)	< 0.0001
Q3	1.83 (1.76, 1.91)	< 0.0001	1.60 (1.54, 1.67)	< 0.0001	1.34 (1.25, 1.43)	< 0.0001
90 mmHg > Diastolic pressure ≥ 80 mmHg						
LHR	1.44 (1.41, 1.47)	< 0.0001	1.34 (1.32, 1.37)	< 0.0001	1.24 (1.20, 1.28)	< 0.0001
Q1	Ref.		Ref.		Ref.	
Q2	1.43 (1.38, 1.48)	< 0.0001	1.33 (1.29, 1.39)	< 0.0001	1.26 (1.19, 1.34)	< 0.0001
Q3	1.89 (1.83, 1.96)	< 0.0001	1.66 (1.60, 1.72)	< 0.0001	1.44 (1.36, 1.53)	< 0.0001

Annotation: Model 1 adjusted for none; Model 2 adjusted for age, smoking status, and drinking status; Model 3 adjusted for FBG, SCr, BUN, ALT, and AST on the basis of Model 2; LDL/HDL ratio analyzed as a continuous variable and in three groups.

Moreover, the analysis of subgroups showcased notable differences in the strength of the association between LHR and hypertension when considering various demographic and behavioral elements. For instance, the stronger correlation found in older age groups aligns with previous research, which emphasizes that factors like gender and age play crucial roles in modifying cardiovascular disease (CVD) risk^[18,19]. Specifically, the association was more significant among females. Conversely, a cross-sectional investigation involving Russian participants indicated that males exhibited considerably higher LHR trajectories compared to females. This variation might stem from the limited sample size and narrow age range of individuals in the Russian research^[20]. Importantly, the heightened sensitivity of LHR observed in those with low BMI and non-smokers raises particular concerns, as such findings are relatively uncommon in existing literature. This implies that even individuals who are usually seen as having a lower CVD risk, such as low BMI individuals and non-smokers, might encounter considerable risk if they present with elevated LHR^[21], highlighting the need for additional studies to validate these observations.

In recent times, there has been a marked focus in China on managing hypertension, highlighted by well-defined objectives and treatment strategies outlined in pertinent health policies. These policies exhibit a notable uniformity in their approaches to hypertension management^[22]. As a result, our research broadens the comprehension of the link between LHR and hypertension. According to established diagnostic guidelines, hypertension is classified when SBP reaches or exceeds 140 mmHg or DBP is at least 90 mmHg, measured on three different occasions without any antihypertensive treatment^[23]. Our findings indicated

that not only were SBP levels of 140 mmHg or more and DBP levels of 90 mmHg or higher closely associated with LHR, but there was also a significant association with SBP from 130 to 140 mmHg and DBP from 80 to 90 mmHg. These results reinforce the potential of LHR as a marker for pre-hypertension.

The current research encompassed a substantial sample size, a varied participant demographic, and thorough data collection methods, all of which improve the applicability of our results. Nonetheless, there are limitations. To begin with, hypertension in the current study relied on a solitary blood pressure reading acquired during a standard health assessment, differing from the established clinical guideline of obtaining three distinct measurements over multiple days. This approach might result in an inaccurate classification of hypertension status, especially among individuals with borderline readings. Additionally, the subgroup analysis for SBP between 130-140 mmHg and DBP from 80-90 mmHg was conducted using just one measurement, which restricts the accuracy of these categorizations. Furthermore, the study was based on retrospective baseline data, potentially overlooking the dynamic or longitudinal connection between LHR and hypertension. Lastly, limitations encountered with the Rich Healthcare database meant that the causes of hypertension (including renal or endocrine factors) could not be fully determined; consequently, we were unable to explicitly rule out instances of secondary hypertension. Future prospective investigations that incorporate repeated blood pressure assessments and more comprehensive clinical data are essential for validating and expanding our conclusions.

CONCLUSION

This research highlights the important connection between the LHR and the prevalence of hypertension in a varied population aged 20 years and older in China. Our results indicate that LHR serves as a strong predictor of hypertension within large populations, with differences noted among various demographic and behavioral groups. The identified threshold in the nonlinear relationship emphasizes the necessity of including LHR in the evaluation of hypertension risk. Future studies should aim to explore the mechanisms that underpin these associations.

DECLARATIONS

Authors' contributions

Conceived and designed research, synthesized and interpreted data, drafted, edited, revised, and approved the final manuscript: Lai W, Chen X, Wang L, Li X, Liu M, Wu L, Zhou B

Availability of data and materials

The data are openly available at www.datadryad.org (10.5061/dryad.ft8750v).

Financial support and sponsorship

This work was supported by the Beijing Municipal Health Commission (2024-3-034) and the tenth cycle (2018-2022) Nanjing Key Medical Specialties.

Conflicts of interest

Dr. Zhou B is a Youth Editorial Board member of *The Journal of Cardiovascular Aging*. The peer review process was conducted independently to ensure objectivity, while the other authors have declared that they have no conflicts of interest.

Ethical approval and consent to participate

The study was approved by the Rich Healthcare Group Review Board, and data were gathered de-identified and retrospectively. Because de-identified information was used, it is not required to obtain patient consent.

The analysis in this study was approved by the Beijing Tsinghua Changgung Hospital Ethical Review Board (25442-4-01), and the requirement for ethical review was waived.

Consent for publication

Not applicable.

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