Association Between Cumulative Lipid Accumulation Index and Hypertension Incidence: A Prospective Cohort Study Postprint

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Abstract

Background Hypertension is a major contributor to global disease burden and mortality, and has become one of the prevalent public health issues in contemporary society. The Cumulative Lipid Accumulation Product (LAP) can reflect the degree of abdominal and visceral fat accumulation. Multiple cross-sectional studies have demonstrated a significant correlation between LAP and hypertension; however, these studies have not adequately considered the impact of long-term exposure on hypertension incidence risk, and research on the causal relationship between LAP and hypertension incidence remains relatively scarce. Objective To evaluate the association between cumulative LAP and the risk of hypertension incidence, and to explore the predictive ability of this indicator for hypertension onset. Methods Data from the Guizhou Natural Population Cohort Study database were utilized. Study participants were enrolled from November 20, 2010 to December 19, 2012, with baseline data collected, and were followed up from April 2016 to October 2020. A total of 3,548 participants were ultimately included. Participants were divided into four levels based on cumulative LAP (Q1-Q4 groups): Q1 group (cumulative LAP $\leq$ 14.54, n=887), Q2 group (14.54 $<$ cumulative LAP $\leq$ 24.35, n=887), Q3 group (24.35 $<$ cumulative LAP $\leq$ 39.21, n=887), and Q4 group (cumulative LAP $>$ 39.21, n=887). Participants were also categorized into non-hypertension group (n=2,696) and hypertension group (n=852) based on the presence of new-onset hypertension. Cox proportional regression models were employed to evaluate the relationship between cumulative LAP and hypertension. Restricted cubic splines were used to assess the dose-response relationship between cumulative LAP and hypertension risk. Time-dependent receiver operating characteristic (ROC) curves for cumulative LAP predicting hypertension were plotted. Participants with follow-up time <3 years and prehypertensive individuals were excluded. Results Among the 3,548 adults included, there were 1,607 males (45.3%) and 1,941 females (54.7%), with

a mean age of (42.5$±$14.1) years. During follow-up, 852 cases (11.2%) were newly diagnosed with hypertension. Comparisons between the non-hypertension and hypertension groups revealed statistically significant differences in gender, age, education level, family history of hypertension, excessive oil intake, excessive salt intake, insufficient fresh fruit intake, sleep deficiency proportion, BMI, diastolic blood pressure, fasting plasma glucose (FPG), and cumulative LAP (P<0.05). Cox proportional regression model results showed that compared with the Q1 group, after adjusting for potential covariates, the risk of hypertension incidence increased progressively in Q2 group (aHR=1.330, 95%CI=1.053-1.681), Q3 group (aHR=1.706, 95%CI=1.364-2.134), and Q4 group (aHR=2.339, 95%CI=1.869-2.928). Restricted cubic spline results demonstrated a non-linear dose-response relationship between cumulative LAP and hypertension risk (P for non-linearity <0.01), with the risk of new-onset hypertension increasing as cumulative LAP increased, but stabilizing after cumulative LAP >65. Time-dependent ROC curves for cumulative LAP predicting new-onset hypertension showed that the area under the ROC curve (AUC) for predicting hypertension incidence in the total population after continuous average exposure of 6, 7, 8, and 9 years was 0.617, 0.590, 0.603, and 0.634, respectively. The AUC for male hypertension incidence was 0.600, 0.561, 0.571, and 0.558, respectively. The AUC for female hypertension incidence was 0.638, 0.629, 0.647, and 0.711, respectively. The AUC for urban population hypertension incidence was 0.596, 0.565, 0.602, and 0.621, respectively. The AUC for rural population hypertension incidence was 0.629, 0.592, 0.594, and 0.635, respectively. Conclusion Cumulative LAP is an independent risk factor for hypertension incidence, but it is not an ideal indicator for predicting hypertension onset, and its predictive value for hypertension incidence is relatively limited.

Full Text

The Association between Cumulative Lipid Accumulation Index and Hypertension: A Prospective Cohort Study

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Abstract

Background

Hypertension remains a major contributor to the global disease burden and mortality, representing a critical public health challenge. While the cumulative lipid accumulation product (LAP), a marker of abdominal and visceral fat deposition, has shown cross-sectional associations with hypertension, existing studies inadequately address the impact of long-term cumulative LAP exposure on hypertension risk. Furthermore, causal relationships between cumulative LAP and incident hypertension remain underexplored.

Objective

To evaluate the association between cumulative LAP and hypertension risk and assess its predictive capacity for incident hypertension.

Methods

Data were derived from the Guizhou Natural Population Cohort Study database. Participants were enrolled from November 20, 2010, to December 19, 2012, and followed up from April 2016 to October 2020. A total of 3,548 subjects were ultimately included in the analysis. Participants were divided into four quartiles based on cumulative LAP (Q1-Q4 groups): Q1 (cumulative LAP $\leq$ 14.54, n=887), Q2 (14.54 $<$ cumulative LAP $\leq$ 24.35, n=887), Q3 (24.35 $<$ cumulative LAP $\leq$ 39.21, n=887), and Q4 (cumulative LAP $>$ 39.21, n=887). Subjects were further categorized into non-hypertension group (n=2,696) and hypertension group (n=852) based on the development of new-onset hypertension. The relationship between cumulative LAP and hypertension was evaluated using the Cox proportional hazards regression model. The dose-response relationship between cumulative LAP and hypertension risk was assessed using restricted cubic splines. Time-dependent receiver operating characteristic (ROC) curves were constructed to evaluate the predictive ability of cumulative LAP for hypertension. Participants with follow-up duration <3 years and those with prehypertension were excluded.

Results

Among the 3,548 adults included, 1,607 (45.3%) were men and 1,941 (54.7%) were women, with a mean age of 42.5 $\pm$ 14.1 years. During the follow-up period, 852 (11.2%) subjects were newly diagnosed with hypertension. Significant differences were observed between the non-hypertension and hypertension groups in terms of gender, age, education level, family history of hypertension, excessive oil intake, excessive salt intake, insufficient fresh fruit intake, proportion of inadequate sleep, body mass index (BMI), diastolic blood pressure, fasting plasma glucose (FPG), and cumulative LAP ($P < 0.05$). The Cox proportional hazards regression model showed that, compared with the Q1 group, the risk of hypertension increased progressively in the Q2 group (aHR = 1.330, 95%CI = 1.053-1.681), Q3 group (aHR = 1.706, 95%CI = 1.364-2.134), and Q4 group (aHR = 2.339, 95%CI = 1.869-2.928) after adjusting for potential confounders. Restricted cubic spline analysis revealed a non-linear dose-response relationship

between cumulative LAP and hypertension risk (Pnon-linearity < 0.01), with the risk of new-onset hypertension increasing with cumulative LAP but stabilizing after cumulative LAP > 65. The time-dependent ROC curves for predicting hypertension incidence showed that the area under the ROC curve (AUC) for the overall population was 0.617, 0.590, 0.603, and 0.634 for continuous average exposure of 6, 7, 8, and 9 years, respectively. The AUC for men was 0.600, 0.561, 0.571, and 0.558, and for women, it was 0.638, 0.629, 0.647, and 0.711. For urban populations, the AUC was 0.596, 0.565, 0.602, and 0.621, while for rural populations, it was 0.629, 0.592, 0.594, and 0.635.

Conclusion
Cumulative LAP is an independent risk factor for the onset of hypertension, but it is not an ideal indicator for predicting the onset of hypertension, and its predictive value for the onset of hypertension is relatively limited.

Keywords
Hypertension; Lipid accumulation product; Cohort study; Forecast

Introduction

Hypertension represents one of the most prevalent public health challenges worldwide and constitutes a major contributor to global disease burden and mortality. According to the Global Burden of Disease Study, approximately one-quarter of adults globally suffer from hypertension, with 9.4 million deaths annually attributed to poor blood pressure control and 212 million disability-adjusted life years lost due to hypertension [1]. As a modifiable risk factor for all-cause mortality, controlling hypertension is crucial for preventing and reducing disease burden and death in populations. Research indicates that obese individuals have 3.5 times higher likelihood of developing hypertension compared to those with normal weight [2,3].

In 2005, Kahn [4] proposed the concept of the lipid accumulation product (LAP), which combines waist circumference (WC) and triglycerides (TG) to simultaneously reflect abdominal and visceral fat accumulation. As research on this index has deepened, multiple cross-sectional studies have demonstrated a significant association between LAP and hypertension, with stronger correlations than other obesity indices [5]. Some studies have also suggested interactive effects between LAP and smoking or family history of hypertension on hypertension risk [6,7]. A 2018 cross-sectional survey based on Chinese populations considered LAP as a potentially important predictor of hypertension [8]. However, these perspectives largely derive from cross-sectional studies that measured LAP at a single time point, failing to adequately consider the impact of long-term exposure and potential changes. This study aims to utilize cohort data to evaluate the relationship between cumulative LAP and hypertension incidence risk and to explore the predictive capacity of this indicator for hypertension development.

Methods

Study Population and Design

This study utilized the Guizhou Natural Population Cohort Study database. Using a multi-stage stratified cluster sampling method, 9,280 permanent residents from 48 townships across 12 districts in Guizhou Province (including 5 urban districts and 7 county towns) were selected for baseline survey between November 20, 2010, and December 19, 2012. Inclusion criteria were: (1) age 18 years or older; (2) residence in the study area for more than 6 months with no plans to relocate; (3) ability to complete questionnaires and blood draws; and (4) signed informed consent. Follow-up surveys were completed for 8,163 participants between April 2016 and October 2020, with 1,117 lost to follow-up (12.04%), yielding a follow-up rate of 87.96%. After excluding 2,057 participants with hypertension history at baseline, 2,421 with missing WC or TG data during follow-up, and 9 with unclear status, a total of 3,548 subjects were included in the final analysis. The study protocol was approved by the Ethics Committee of Guizhou Provincial Center for Disease Control and Prevention (S2017-02). The participant selection process is illustrated in [Figure 1: see original paper].

Data Collection

Baseline information was collected using the Chronic Disease and Risk Factor Surveillance Questionnaire for Chinese Adults, designed by the Chinese Center for Disease Control and Prevention. Trained medical staff conducted face-to-face interviews to gather demographic characteristics (gender, age, residential area, education level, marital status, occupation), lifestyle factors (smoking, alcohol consumption, physical activity), and major chronic disease history. Follow-up surveys were conducted from December 2016 to June 2020 using the same methods and content as baseline. The endpoint event was defined as incident hypertension during follow-up, while censoring events included loss to follow-up, no hypertension by study end, or missing data. Follow-up person-years were calculated from enrollment until hypertension diagnosis, death, or final follow-up, whichever occurred first.

Measurements and Laboratory Tests

During baseline and follow-up surveys, trained measurers used standardized instruments to measure height, weight, waist circumference, and blood pressure. Blood pressure was measured three times at 1-minute intervals. If the three measurements differed by ≤ 10 mmHg (1 mmHg = 0.133 kPa), the average of the three values was used as the final reading. If measurements differed substantially, the average of the two most similar values was used. Waist circumference was measured at the midpoint between the lowest rib margin and the iliac crest, recorded to 0.1 cm, with the average of three measurements used as the final value. Height and weight were similarly averaged across three measurements. After at least 8 hours of fasting, venous blood samples were collected to measure

fasting plasma glucose (FPG), total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). All blood samples were sent to the Guizhou Provincial Center for Disease Control and Prevention for uniform testing.

Definition of Hypertension

Hypertension was defined as systolic blood pressure (SBP) $\geq$ 140 mmHg and/or diastolic blood pressure (DBP) $\geq$ 90 mmHg [9], or use of antihypertensive medication, or physician diagnosis, or self-reported hypertension. Pre-hypertension was defined as SBP = 130–139 mmHg (1 mmHg = 0.133 kPa) and/or DBP = 80–89 mmHg.

Calculation of Cumulative LAP

Cumulative LAP was calculated using the following formulas:
For men: LAP = [WC (cm) -65] $\times$ TG (mmol/L)
For women: LAP = [WC (cm) -58] $\times$ TG (mmol/L)
Cumulative LAP = (baseline LAP + follow-up LAP) / 2, representing the average LAP level under long-term exposure. Based on cumulative LAP quartiles, participants were divided into four levels (Q1–Q4 groups): Q1 (cumulative LAP $\leq$ 14.54, n = 887), Q2 (14.54 $<$ cumulative LAP $\leq$ 24.35, n = 887), Q3 (24.35 $<$ cumulative LAP $\leq$ 39.21, n = 887), and Q4 (cumulative LAP $>$ 39.21, n = 887).

Statistical Analysis

Statistical analysis was performed using R version 4.3.2. The Shapiro-Wilk test was used to assess normality. Normally distributed continuous variables were expressed as mean $\pm$ standard deviation and compared between groups using independent samples t-tests; non-normally distributed variables were expressed as median (P25, P75) and compared using rank-sum tests. Categorical variables were expressed as frequencies (percentages) and compared using ^{2} tests. Cox proportional hazards regression models were used to evaluate the relationship between cumulative LAP and hypertension, with adjustment for covariates. Restricted cubic splines were used to assess the dose-response relationship between cumulative LAP and hypertension risk. Time-dependent receiver operating characteristic (ROC) curves were constructed to evaluate the predictive ability of cumulative LAP for hypertension. Sensitivity analyses were performed by excluding participants with follow-up duration $<$ 3 years, those with pre-hypertension, and by randomly selecting 50% and 40% of the study population. All statistical tests were two-sided, with $P < 0.05$ considered statistically significant.

Results

Baseline Characteristics

A total of 3,548 adults were included in the analysis, comprising 1,607 men (45.3%) and 1,941 women (54.7%), with a mean age of 42.5 ± 14.1 years. The mean FPG was 5.22 ± 1.25 mmol/L, mean SBP was 116 ± 12 mmHg, and mean DBP was 74 ± 8 mmHg. The average follow-up duration was 6.81 ± 1.34 person-years, during which 852 participants (11.2%) were newly diagnosed with hypertension. A family history of hypertension was present in 398 participants (11.2%), and 2,855 (80.5%) were married or cohabitating. Urban residents accounted for 1,279 participants (36.0%). Participants were divided into non-hypertension (n = 2,696) and hypertension (n = 852) groups based on incident hypertension status. Significant differences between groups were observed for gender, age, education level, family history of hypertension, excessive oil intake, excessive salt intake, insufficient fresh fruit intake, proportion of inadequate sleep, BMI, DBP, FPG, and cumulative LAP ($P < 0.05$). No significant differences were found for residential area, marital status, physical activity, smoking, alcohol consumption, dyslipidemia, insufficient grain and starch intake, insufficient fresh vegetable intake, excessive red meat intake, or SBP ($P > 0.05$). Detailed baseline characteristics are presented in .

Cumulative LAP and Incident Hypertension Risk

The incidence densities for Q1–Q4 groups were 20.34, 27.83, 40.90, and 52.23 per 1,000 person-years, respectively. Using Cox proportional hazards regression with new-onset hypertension (yes = 1, no = 0) as the dependent variable and cumulative LAP quartile group (Q1 = 1, Q2 = 2, Q3 = 3, Q4 = 4) as the independent variable, the results showed that each unit increase in cumulative LAP was associated with increased hypertension risk after adjusting for gender, age, education level, family history of hypertension, BMI, SBP, DBP, FPG, excessive oil intake, excessive salt intake, insufficient fresh fruit intake, and inadequate sleep. Compared with Q1, the adjusted hazard ratios (aHR) were 1.330 (95%CI = 1.053–1.681) for Q2, 1.706 (95%CI = 1.364–2.134) for Q3, and 2.339 (95%CI = 1.869–2.928) for Q4, with a significant trend across quartiles ($P_{trend} < 0.05$). These results are summarized in .

Dose-Response Relationship

Restricted cubic spline analysis was used to evaluate the dose-response relationship between cumulative LAP and hypertension risk. After adjusting for gender, age, education level, family history of hypertension, BMI, SBP, DBP, FPG, excessive oil intake, excessive salt intake, insufficient fresh fruit intake, and inadequate sleep, cumulative LAP showed a non-linear dose-response relationship with hypertension risk ($P_{non\text{-}linearity} < 0.01$). The risk of new-onset hypertension increased with cumulative LAP but plateaued when cumulative LAP exceeded 65. This pattern was consistent across different gender groups,

as illustrated in [Figure 2: see original paper].

Predictive Value of Cumulative LAP

Time-dependent ROC curves were constructed to assess the predictive value of cumulative LAP for new-onset hypertension. The area under the ROC curve (AUC) for the overall population was 0.617, 0.590, 0.603, and 0.634 for continuous average exposures of 6, 7, 8, and 9 years, respectively. For men, the AUC values were 0.600, 0.561, 0.571, and 0.558; for women, they were 0.638, 0.629, 0.647, and 0.711. In urban populations, the AUC values were 0.596, 0.565, 0.602, and 0.621, while in rural populations they were 0.629, 0.592, 0.594, and 0.635. These results are presented in and [Figure 3: see original paper].

Sensitivity Analysis

Sensitivity analyses were performed by excluding participants with follow-up duration < 3 years, those with pre-hypertension, and by randomly selecting 50% and 40% of the study population. The results remained consistent with the main findings, as shown in [Figure 4: see original paper].

Discussion

Numerous studies have demonstrated that visceral fat distribution is closely associated with pre-hypertension and cardiovascular disease, with more detrimental cardiometabolic effects than subcutaneous fat [9–11]. Excess visceral adipose tissue may lead to lipotoxicity, insulin resistance, and increased inflammatory mediators [12,13]. Visceral fat accumulation is associated with insulin resistance, which may accelerate the progression of hypertension and atherosclerosis and stimulate blood pressure elevation [14]. Triglycerides, as an indicator of visceral fat accumulation, represent an established risk factor for hypertension. Furthermore, extensive research has shown that waist circumference, as an indicator of abdominal obesity and a predictor of numerous chronic diseases, is significantly associated with elevated blood pressure [15,16], with each 1 cm increase in WC associated with a 1.2-fold increase in hypertension risk [17,18]. Both elevated TG and increased WC are independent risk factors for hypertension [19–24].

In 2005, Kahn [4] proposed the LAP concept to assess lipid overaccumulation in adults. This novel index combines the anatomical indicator WC with the physiological indicator TG, demonstrating superior identification of diabetes and cardiovascular disease risk compared to BMI and better reflecting visceral lipid accumulation. Since its introduction, increasing research has examined the relationship between LAP and various diseases. Cross-sectional studies have shown significant associations between LAP and hypertension [5–8]. However, these studies used LAP measurements at a single time point and did not fully consider the effects of long-term exposure and potential changes. Measuring cumulative LAP can provide more comprehensive insight into hypertension risk associated with changes in body fat distribution [6]. Nonetheless, these cross-sectional

investigations cannot establish causality. To date, few studies have examined the causal relationship between LAP and hypertension incidence. Our study addresses this gap through long-term follow-up, investigating the relationship between continuous LAP exposure and new-onset hypertension. Our findings further confirm that LAP is a risk factor for hypertension incidence, showing a non-linear dose-response relationship with hypertension risk. Within a certain range, LAP level is positively correlated with hypertension risk, with the adjusted hazard ratio increasing significantly as LAP index rises, though the risk stabilizes when cumulative LAP exceeds 65.

Our study found that cumulative LAP has limited predictive capacity for new-onset hypertension, with modest accuracy and reliability, making it less than ideal for predicting hypertension incidence in this adult population. These findings differ from some previous studies. For example, a 2020 cross-sectional survey of 683 Chinese children and adolescents aged 8–15 years suggested that childhood LAP could serve as a novel indicator for predicting pediatric hypertension [25]. A 2013 study of 2,589 Mongolian adults in China proposed that LAP was more closely associated with hypertension risk than BMI in Mongolian men, suggesting LAP as a potential predictor of hypertension risk in males [26]. The 2023 China Health and Retirement Longitudinal Study showed that LAP had moderate predictive value for hypertension among adults aged 45 years and older (AUC = 0.579 for men with 47.60% sensitivity and 67.30% specificity; AUC = 0.594 for women with 72.50% sensitivity and 44.00% specificity) [27]. A 2018 community-based cross-sectional study in Bengbu, China, found that elevated LAP was significantly associated with higher hypertension risk among Han Chinese adults [5]. Another 2019 cross-sectional survey of 2,079 community adults in southern China also demonstrated a significant association between LAP and hypertension risk.

The LAP measures in these studies were derived from cross-sectional data or baseline measurements from longitudinal studies. In contrast, our study incorporated both baseline and follow-up LAP data, reflecting long-term exposure patterns. The discrepancies in findings may be attributable to differences in study methodology and populations, warranting further investigation.

Our study employed a prospective cohort design with long-term follow-up and comprehensive data on lifestyle factors and potential confounders. By utilizing both baseline and follow-up LAP values, our analysis better captures the effects of long-term exposure, providing robust evidence for the causal relationship between LAP and hypertension incidence. However, several limitations should be acknowledged. First, the sample size was relatively modest, and larger-scale or longer-term studies are needed. Second, the study population was limited to adults from a specific region, which may limit generalizability to other populations. Additionally, certain potential confounders such as genetic variations were not addressed, which may have influenced the results.

In summary, our findings indicate that cumulative LAP is an independent risk factor for hypertension incidence but is not an ideal predictor of new-onset

hypertension. Further research is needed to identify more effective predictive indicators to provide scientific evidence for early identification and management of hypertensive patients.

Author Contributions: MEI Jingyan conceived the study, designed the research, and wrote the manuscript; CHEN Min and ZHANG Lieqiang collected and organized data and provided statistical methodology; PAN Yunxi and WANG Xin performed statistical analyses and created figures; ZHAO Xiaodeng and ZHAN Wei revised the manuscript; LIU Tao and WANG Yiying provided guidance, supervised quality control, and oversaw the project.

Conflict of Interest: The authors declare no conflicts of interest.

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Note: Figure translations are in progress. See original paper for figures.