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Gender-specific thresholds and nonlinear associations of obesity indices with hyperlipidemia: insights from NHANES 2007–2020

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Abstract. Hyperlipidemia, a significant contributor to cardiovascular ailments, have a close association with obesity, is closely linked to obesity. Traditional anthropometric indices (BMI, WHR, WHtR) are widely used to assess obesity, but their nonlinear relationships with hyperlipidemia and gender-specific risk thresholds remain poorly understood. Leveraging data from 2,739 participants in the NHANES 2007–2020, this research utilized multivariable logistic regression analysis along with restricted cubic splines (RCS) to investigate both linear and nonlinear relationships between various obesity indices and the presence of hyperlipidemia. Nonlinear dose-response relationships were observed for BMI, WHR, and WHtR with hyperlipidemia (*P* for nonlinear < 0.001). Gender-specific risk thresholds were identified: males: WHR > 0.94, WHtR > 0.76, BMI >28.0; Females: WHR 0.91–0.99, WHtR 0.61–0.69, BMI 29.4–34.39. Gender differences in effect sizes were significant (e.g., WHR β: males 6.986, *P*<0.001 vs. females 5.666, *P*=0.023). Subgroup analysis showed stronger associations in younger individuals (≤50 years) without hypertension or diabetes. WHR remained an independent predictor in metabolic comorbidities, while BMI and WHtR were confounded by metabolic disturbances. This study highlights the importance of gender-specific risk thresholds for obesity indices, particularly WHR, in predicting hyperlipidemia. The results demonstrate that WHR serves as a more reliable predictor for metabolic disorder risks compared to other anthropometric indices, particularly highlighting the importance of implementing preventive measures in populations under 50 years of age. Future research should focus on developing precision stratification models to optimize obesity management.

Keywords: hyperlipidemia, obesity indices, Body Mass Index (BMI), Waist-to-Hip Ratio (WHR), Waist-to-Height Ratio (WHtR)

1. Introduction

Hyperlipidemia is a major contributor to cardiovascular disease and has become increasingly prevalent worldwide, creating a substantial public health challenge [1]. Research indicates that obesity and related metabolic imbalances are strongly linked to dyslipidemia [2]. Common measures Like Body Mass Index (BMI), Waist-to-Hip Ratio (WHR), and Waist-to-Height Ratio (WHtR) are widely used to evaluate general and abdominal obesity, though their clinical effectiveness warrants further investigation [3]. Although BMI is a standard indicator of overall obesity, it does not differentiate fat distribution, especially visceral adiposity, which is more closely associated with metabolic and cardiovascular complications [4, 5]. On the other hand, WHR and WHtR offer more accurate assessments of abdominal obesity by accounting for fat accumulation around the midsection—a critical factor in hyperlipidemia and cardiovascular risk [6, 7]. Nevertheless, whether WHR and WHtR independently predict lipid metabolism disorders remains debated [8]. Additionally, earlier research has mostly assumed linear relationships, overlooking potential nonlinear trends (e.g., dose-response effects) between body measurements and disease likelihood, possibly resulting in skewed risk evaluations and suboptimal clinical thresholds.

In recent years, the application of nonlinear association analysis in epidemiological research has gained prominence. Some scholars have proposed that the impact of BMI on hyperlipidemia may follow a “J-shaped” curve, where risk remains relatively stable below a certain threshold but increases exponentially beyond it. Research suggests a nonlinear dose-response association between BMI and hyperlipidemia, where each 1 kg/m² increase in BMI corresponds to a marked elevation in hyperlipidemia risk [9]. However, research on WHR and WHtR has largely been confined to linear association analyses, with insufficient exploration of their nonlinear effects [10, 11]. Therefore, identifying risk inflection points through nonlinear models such as Restricted Cubic Splines (RCS) may provide critical insights for optimizing clinical decision-making.

Sex-specific variations significantly influence the association between obesity and hyperlipidemia. Studies suggest sexual dimorphism in fat distribution, with males exhibiting higher visceral adiposity (linked to dyslipidemia and insulin resistance) compared to females who preferentially store subcutaneous fat - a pattern that confers relative metabolic protection until menopausal hormonal changes occur [12, 13]. These differences highlight the need for gender-specific analyses when evaluating the predictive power of anthropometric indices.

This study, based on large-scale cross-sectional data, integrates linear and nonlinear analytical frameworks to comprehensively examine the association patterns of BMI, WHR, and WHtR with hyperlipidemia. The findings are expected to contribute to dynamic risk assessment tools for early screening of hyperlipidemia and facilitate more effective prevention strategies in high-risk populations.

2. Methods

2.1. Data source and study population

This research utilized data collected from the National Health and Nutrition Examination Survey (NHANES) across cycles spanning 2007 to 2020. NHANES participant recruitment and data collection procedures underwent rigorous review and received ethical approval from an institutional review board. Written informed consent was obtained from all participants prior to their involvement, ensuring autonomy, confidentiality, and adherence to ethical standards.

Participants aged 20 years or older were included in the study. Eligibility criteria required complete data on waist circumference, height, hip circumference, and lipid profiles. Individuals with liver or thyroid disorders, malignancies, or those receiving hormonal treatments (e.g., corticosteroids, sex hormones, or parathyroid hormones) were excluded to ensure sample representativeness and data accuracy. Ultimately, 2,739 eligible participants were included in the study. The participant selection and exclusion process are illustrated in Figure 1.

2.2. Measurement of waist circumference, hip circumference, and lipid profiles

WHR was calculated as waist/hip circumference, WHtR as waist/height, and BMI as weight(kg)/height^2(m^2). All measurements were recorded in centimeters except BMI (kg/m^2). Participants fasted for 9-12 hours prior to venipuncture blood collection, with samples analyzed using enzymatic methods (Cobas 6000) for TC and TG measurement. LDL-C was calculated via the validated Friedewald equation (LDL-C=TC-HDL-C-TG/5; r=0.89 versus direct measurement), while HDL-C was measured directly [14]. Hyperlipidemia was defined as meeting any of the following criteria: TC ≥200 mg/dL, TG ≥150 mg/dL, HDL-C <40 mg/dL, LDL-C ≥130 mg/dL, or current use of lipid-lowering medications (within 30 days) [15].

2.3. Covariates

Based on previous studies [9, 16]. This study examined demographic and clinical variables including age, sex, ethnicity (Mexican American, other Hispanic, non-Hispanic White, non-Hispanic Black, and other races), education level (<high school, high school, >high school), hypertension and diabetes history (self-reported physician-diagnosed), smoking status (non-smoker: <100 lifetime cigarettes; smoker: ≥100 cigarettes), and alcohol consumption (non-drinker: ≤12 lifetime drinks; moderate: ≤2 drinks/day for men or ≤1 for women; heavy: ≥3 drinks/day for men or ≥2 for women). Data were collected via the health history questionnaire.

2.4. Statistical analysis

NHANES used a carefully designed sampling approach with weighting adjustments to ensure the results represent the U.S. population. We summarized key characteristics of the participants. Normally distributed data are presented as weighted means ± standard deviations, non-normal data as medians (interquartile ranges), and categorical variables as weighted percentages.

A multivariable logistic regression analysis was conducted to examine the associations between BMI, WHR, WHtR, and hyperlipidemia. Three separate models were developed to represent the entire population, as well as distinct subgroups for males and females, [1] an unadjusted model, [2] a partially adjusted model (taking into account age and education level, and ethnicity), along with [3] a completely adjusted model.

RCS curves were utilized to test for nonlinear relationships and visualize dose-response associations under the fully adjusted model with weighting. The RCS curves were constructed with five knots placed at the 5th, 27.5th, 50th, 72.5th, and 95th percentiles as determined by the Akaike Information Criterion (AIC) for optimal model fit [17, 18]. Subgroup analysis was performed to explore the correlations among BMI, WHR, and WHtR. and hyperlipidemia in various population groups, such as age, presence of hypertension, diabetes history, and smoking habits. The influence of interaction between grouping factors and independent variables on the dependent variable was examined. The outcomes of the subgroup analyses were represented visually through forest plots.

All statistical analyses were performed using R software (version 4.3.1; R Foundation for Statistical Computing, Vienna, Austria).

3. Results

A total of 2,739 participants were ultimately included in the study. Compared to the excluded population, the included participants had a higher proportion of males (55.46% vs. 47.46%, $P < 0.001$) and a higher BMI (29.48 vs. 28.36, $P < 0.001$). However, there was no significant difference in age between the included and excluded populations (42.99 vs. 42.84, $P = 0.051$). As shown in **Table 1**, significant differences were observed between males and females in terms of BMI, poverty-to-income ratio (PIR), WHR, WHtR, racial distribution, prevalence of hyperlipidemia, prevalence of diabetes, alcohol consumption, and smoking behavior (all $P < 0.05$). Males had a significantly lower BMI (28.86 ± 0.28 kg/m²) compared to females (30.25 ± 0.37 kg/m², $P = 0.002$), while males had a significantly higher WHR (0.96 ± 0.00) than females (0.89 ± 0.00, $P < 0.001$). Additionally, the weighted prevalence of hyperlipidemia (60.75% vs. 54.06%), diabetes (13.54% vs. 9.65%), and smoking (48.57% vs. 33.49%) was significantly higher in males than in females (all $P < 0.05$).

3.1. Associations of BMI, WHR, and WHtR with hyperlipidemia

Table 2 presents the strength of associations between WHR, WHtR, BMI, and hyperlipidemia based on weighted logistic regression models. In the unadjusted Model 1, WHR ($\beta = 8.805$, $P < 0.001$), WHtR ($\beta = 4.877$, $P < 0.001$), and BMI ($\beta = 0.043$, $P < 0.001$) were all significantly positively associated with hyperlipidemia. After adjusting for age, gender, race, and education level (Model 2), the effect sizes of these indices decreased but remained statistically significant (WHR: $\beta = 6.826$, $P < 0.001$; WHtR: $\beta = 4.058$, $P < 0.001$; BMI: $\beta = 0.049$, $P < 0.001$). Further adjustment for income, hypertension, diabetes, smoking, and alcohol consumption (Model 3) revealed that WHR ($\beta = 6.136$, $P < 0.001$), WHtR ($\beta = 3.753$, $P < 0.001$), and BMI ($\beta = 0.049$, $P < 0.001$) remained independent risk factors for hyperlipidemia, indicating that their associations were not confounded by metabolic or behavioral factors.

Gender-stratified analysis showed that the effect size of WHR was significantly higher in males than in females (Model 3: males $\beta = 6.986$, $P < 0.001$ vs. females $\beta = 5.666$, $P = 0.023$). Similarly, the gender difference in WHtR was significant (males $\beta = 5.556$, $P<0.001$ vs. females $\beta = 2.925$, $P = 0.002$). The association strength of BMI was also higher in males (males $\beta = 0.077$, $P = 0.002$ vs. females $\beta = 0.037$, $P = 0.005$), suggesting that the impact of abdominal fat distribution and overall obesity on hyperlipidemia exhibits gender heterogeneity.

3.2. Nonlinear associations of BMI, WHR, and WHtR with hyperlipidemia

The RCS model was used to analyze the nonlinear associations of BMI, WHR, and WHtR with hyperlipidemia risk (Figure 2, Supplemental Figure 1, Supplemental Figure 2). The results demonstrated significant nonlinear dose-response relationships between these anthropometric indices and hyperlipidemia risk in the overall population and gender subgroups (P for nonlinearity < 0.001). Specifically, WHR exhibited a threshold effect: in the overall population, hyperlipidemia risk significantly increased when WHR exceeded 0.94 (Figure 2). In males, risk similarly increased when WHR exceeded 0.94 (Supplemental Figure 1), while in females, WHR between 0.91 and 0.99 was positively associated with hyperlipidemia risk (Supplemental Figure 2). For WHtR, a positive association with hyperlipidemia risk was observed in the overall population when WHtR ranged from 0.59 to 0.67, indicating that risk increased progressively within this range (Figure 2). In males, risk significantly increased when WHtR exceeded 0.76 (Supplemental Figure 1), while in females, WHtR between 0.61 and 0.69 was positively associated with hyperlipidemia risk (Supplemental Figure 2). For BMI, a positive association with hyperlipidemia risk was observed in the overall population when BMI ranged from 28.6 to 32.69, indicating that risk increased significantly within this range (Figure 2). In males, BMI between 28.00 and 30.48 was positively associated with hyperlipidemia risk, and risk significantly increased when BMI exceeded 39.65 (Supplemental Figure 1). In females, BMI between 29.40 and 34.39 was positively associated with hyperlipidemia risk (Supplemental Figure 2).

3.3. Subgroup analysis of BMI, WHR, and WHtR with hyperlipidemia

Subgroup analysis revealed significant differences in the associations of BMI, WHR, and WHtR with hyperlipidemia risk across different populations (Figure 3). In individuals aged ≤ 50 years, the associations of WHR, WHtR, and BMI with hyperlipidemia risk were statistically significant (all $P < 0.05$), whereas no significant associations were observed in individuals aged > 50 years. For hypertension history, the association of WHR was significant in both hypertensive and non-hypertensive groups, while the associations of WHtR and BMI were significant only in the non-hypertensive group. For diabetes history, the associations of WHR, WHtR, and BMI were significant only in the non-diabetic group. Additionally, smoking status showed significant effects across all subgroups. These results suggest that the associations of BMI, WHR, and WHtR with hyperlipidemia risk are more pronounced

in younger individuals without hypertension or diabetes, indicating that interventions targeting these populations may be more effective.

4. Discussion

This study, based on data from the NHANES, systematically revealed the nonlinear association patterns of BMI, WHR, and WHtR with hyperlipidemia risk. The findings not only confirmed the clinical value of these indices as independent risk factors but also quantified risk thresholds through dose-response curves, providing valuable insights for the precise prevention and control of obesity-related metabolic abnormalities.

From a pathophysiological perspective, this study elucidated the underlying connections between fat distribution indices and lipid abnormalities. Notably, hyperlipidemia risk increased when WHR exceeded 0.94 (in males) or fell within the range of 0.91–0.99 (in females), when WHtR reached 0.76 (in males) or 0.61–0.69 (in females), and when BMI surpassed 28.6 (in males) or 29.4 (in females). This nonlinear association may stem from the dynamic endocrine regulation of adipose tissue. Excessive accumulation of visceral fat triggers a cascade release of pro-inflammatory factors (e.g., TNF-α, IL-6, MCP-1), which act through two primary pathways: 1) activation of inflammatory pathways leading to insulin resistance, thereby disrupting lipoprotein metabolism [19]; and 2) imbalance in adipokines, resulting in a disproportionate ratio of adiponectin to leptin, which promotes excessive hepatic lipid synthesis and ultimately leads to abnormal lipid profiles [20].

Gender specificity emerged as one of the core findings of this study. The effect sizes of WHR (β = 6.986 vs. 5.666), WHtR (β = 5.556 vs. 2.925), and BMI (β = 0.077 vs. 0.037) were significantly higher in males than in females. This disparity may stem from three biological factors: First, males are more prone to visceral obesity, with adipocytes exhibiting higher lipolytic activity and greater secretion of inflammatory factors [21]. Second, estrogen enhances adipocyte differentiation capacity by modulating the PPARγ signaling pathway, thereby reducing the risk of ectopic lipid deposition in females [22]. Third, gender differences in androgen levels may influence lipid metabolism by regulating lipoprotein lipase activity [23, 24]. Additionally, postmenopausal women may experience a masculinized shift in fat distribution [25], underscoring the need for dynamic risk assessment across the lifespan.

The nonlinear models provided in-depth insights into the clinical thresholds of anthropometric indices and their gender-specific variations. For males, high-risk intervals were identified when WHR exceeded 0.94, WHtR surpassed 0.76, or BMI was greater than 28. In contrast, the risk thresholds for females were more complex, encompassing WHR values of 0.91–0.99, WHtR values of 0.61–0.69, and BMI values of 29.4–34.39. These differences not only highlight the importance of gender-specific risk assessment but also offer two key implications for clinical practice: 1) for males, stricter and more precise body shape management is required; and 2) for females, more nuanced and individualized criteria are needed in the evaluation of central obesity.

Subgroup analysis revealed important effect modifiers. In individuals aged ≤ 50 years, all anthropometric indices showed significant associations with hyperlipidemia risk, whereas these associations weakened in those aged > 50 years. This may be related to age-related muscle loss and fat redistribution [26]. Notably, among individuals with hypertension or diabetes, WHR retained its independent predictive value, while the effects of WHtR and BMI were partially masked by metabolic disturbances. This may be attributed to the superior ability of WHR to reflect visceral fat distribution, which is a key driver of metabolic abnormalities [27, 28]. In contrast, BMI and WHtR are more susceptible to confounding factors such as muscle mass loss, edema, or height changes, particularly in populations with metabolic comorbidities [29]. This suggests that WHR should be prioritized for risk assessment in patients with metabolic comorbidities.

This study has both strengths and limitations. Its strengths include using NHANES data (a large U.S. national survey), combining different analysis methods to better understand body measurements and hyperlipidemia links, and finding gender-specific risk levels. However, the study can't prove cause-and-effect due to its design, results may not apply to non-U.S. populations, and it didn't include newer body fat measurement methods that might provide more details.

5. Conclusions and future perspectives

By establishing nonlinear dose-response relationships between anthropometric indices and hyperlipidemia, this study provides quantitative evidence for the precise prevention and control of obesity-related metabolic diseases. The findings highlight the importance of the following in clinical practice: 1) adopting gender-specific risk assessment thresholds, 2) implementing early body shape interventions in younger populations, and 3) prioritizing WHR as a core monitoring indicator for patients with metabolic comorbidities. Future research could focus on developing intelligent risk prediction models and exploring gene-environment interactions to advance obesity management from a "single BMI-oriented approach" to a "multidimensional precision stratification" model.

Data Availability*: All data are available on the NHANES website (https://wwwn.cdc.gov/nchs/nhanes/default.aspx).

Conflict of Interest: Yaning Tong, Liang Ma and Fangzhu Xiao declare that they have no conflict of interest.

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Fangzhu Xiao); final approval of the version to be published (all authors); and agreement to be accountable for all aspects of the work (all authors).

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Appendix

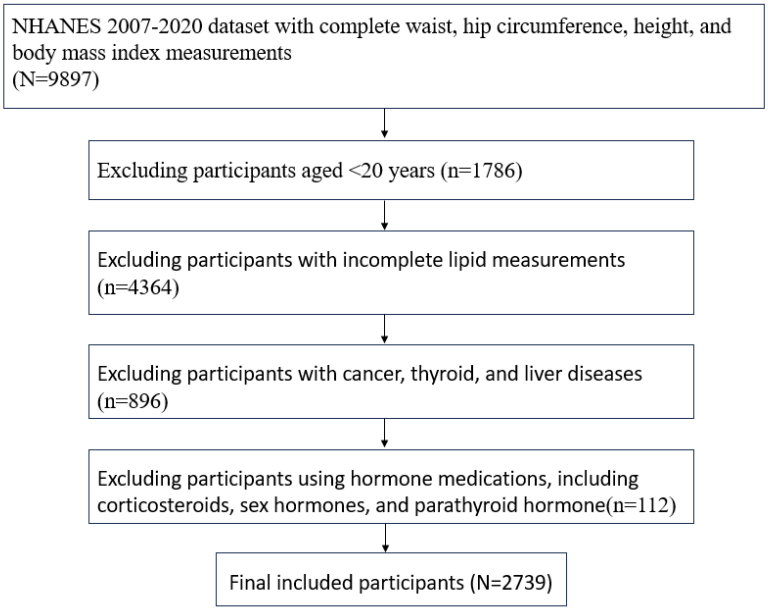


Figure 1. Inclusion exclusion flowchart

Table 1. Weighted basic characteristics of the participants

Variables	Overall N=2739	Male N=1446	Female N=1293	*P* between sex
Age (years)	42.99 (30.27, 56.90)	42.87 (30.13, 56.99)	43.13 (30.45, 56.83)	0.675
Body mass index (kg/m2)	29.48±0.25	28.86±0.28	30.25±0.37	0.002
PIR	3.10 (1.59, 4.98)	3.42 (1.77, 4.98)	2.74 (1.44, 4.98)	0.009
WHR	0.93±0.00	0.96±0.00	0.89±0.00	< 0.001
WHtR	0.59±0.00	0.58±0.00	0.61±0.01	< 0.001
Race, n (weighted %)				0.001
Mexican American	10.64	11.36	9.75	
Other Hispanic	6.90	6.21	7.77	
Non-Hispanic White	58.43	60.36	56.02	
Non-Hispanic Black	13.33	11.46	15.66	
Other Race	10.69	10.61	10.80	
Educational level, n (weighted %)				0.222
< High school	11.28	12.40	9.90	
High school	25.95	26.47	25.31	
> High school	62.77	61.14	64.80	
Hyperlipidemia, n (weighted %)				0.017
Yes	57.77	60.75	54.06	
No	42.23	39.25	45.94	
Hypertension, n (weighted %)				0.075
Yes	28.55	30.11	26.60	
No	71.45	69.89	73.40	
Diabetes, n (weighted %)				< 0.001
Yes	11.81	13.54	9.65	

Table 1. Continued

No	88.19	86.46	90.35	
Alcohol intake, n (weighted %)				< 0.001
Never	7.57	4.98	10.91	
Moderate	45.25	55.60	31.97	
Heavy	47.17	39.43	57.12	
Smoking, n (weighted %)				< 0.001
Yes	41.85	48.57	33.49	
No	58.15	51.43	66.51	

Weighted characteristics of the study population based on WHR quartiles. Continuous variables are expressed as mean ± SD, and P values were calculated from weighted linear regression models; Categorical variables are expressed as n (weight%), and P values were calculated by weighted chi-square tests.

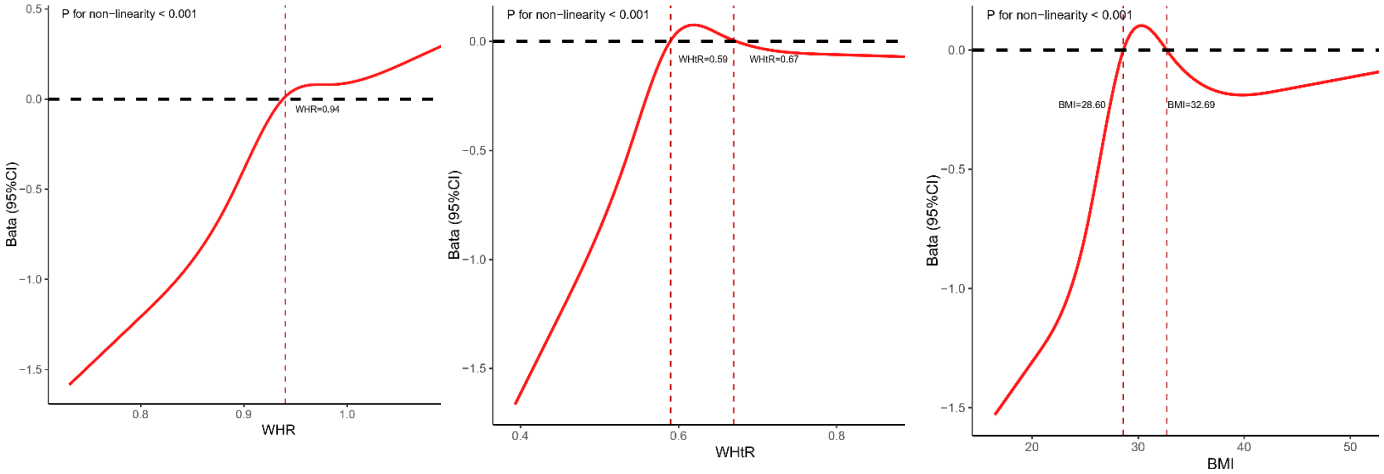
Table 2. Association of waist-to-hip ratio, waist-to-height ratio, BMI and hyperlipidemia

Variables	Overall		Male		Female	
	β	*P*	*β*	*P*	*β*	*P*
			Model 1			
WHR	8.805	< 0.001	11.314	< 0.001	8.246	< 0.001
WHtR	4.877	< 0.001	8.112	< 0.001	3.455	< 0.001
BMI	0.043	< 0.001	0.086	< 0.001	0.023	0.041
			Model 2			
WHR	6.826	< 0.001	8.785	< 0.001	5.790	0.002
WHtR	4.058	< 0.001	6.371	< 0.001	2.869	0.004
BMI	0.049	< 0.001	0.082	< 0.001	0.033	0.004
			Model 3			
WHR	6.136	< 0.001	6.986	< 0.001	5.666	0.023
WHtR	3.753	< 0.001	5.556	0.002	2.925	0.012
BMI	0.049	< 0.001	0.077	0.002	0.037	0.005

Model 1: Unadjusted for covariates.

Model 2: Adjusted for partial covariates (age, education level, race, and sex).

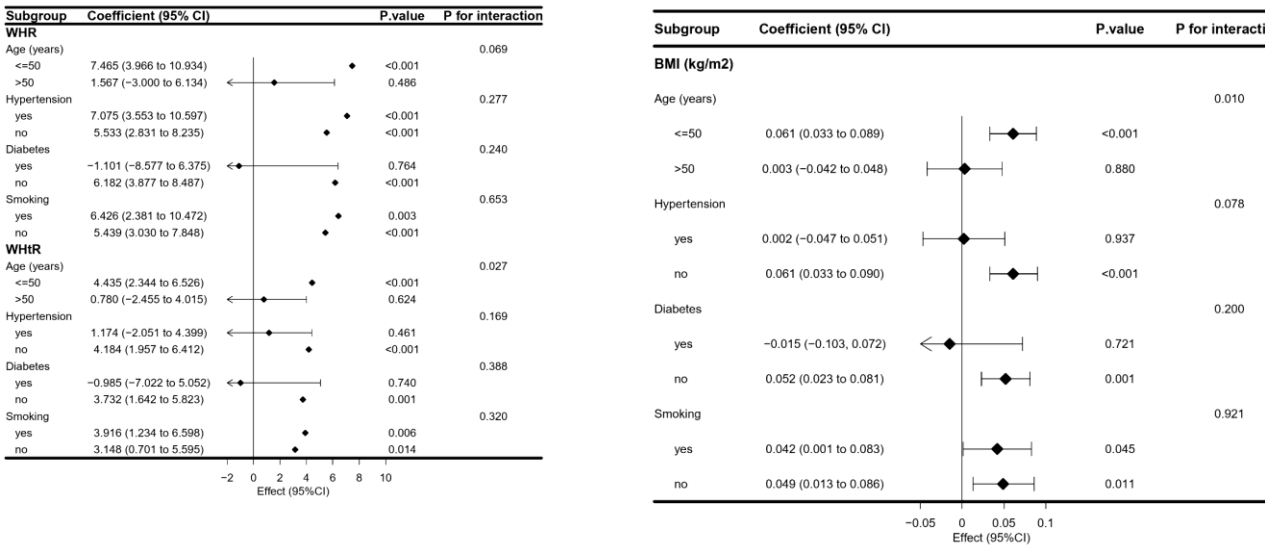
Model 3: Fully adjusted for all covariates (age, sex, race, education level, PIR, history of hypertension, history of diabetes, smoking, and alcohol intake).



(a) (b) (c)

Figure 2. Non-linear association between anthropometric indices (BMI, WHR, and WHtR) and hyperlipidemia

The figures were fully adjusted for all covariates (age, sex, race, education level, PIR, history of hypertension, history of diabetes, smoking, and alcohol intake). The restricted cubic spline (RCS) curve was constructed with 5 knots placed at the 5th, 27.5th, 50th, 72.5th, and 95th percentiles.



(a) (b)

Figure 3. Forest plot of subgroup analysis

The figures were fully adjusted for all covariates (age, sex, race, education level, PIR, history of hypertension, history of diabetes, smoking, and alcohol intake), except for the subgroup variables.