

# Correlation Between the Levels of Fasting and Postprandial ApoB48 with Metabolic Syndrome: A Cross-Sectional Study

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**Purpose:** This study aims to determine apolipoprotein B-48 (ApoB48) levels before and after a high-fat diet in individuals with metabolic syndrome (MetS), and to explore its relationship with MetS. It provides new risk markers for exploring the mechanism of MetS and offers important theoretical basis and candidate targets for subsequent research.

**Patients and Methods:** A total of 192 adult Chinese volunteers were enrolled in this cross-sectional study. Participants were divided into control and MetS groups according to the NCEP ATP III diagnostic criteria for MetS. All participants underwent oral fat tolerance testing (OFTT). Serum concentrations of fasting and postprandial ApoB48 were measured, and their relationships with each MetS component were analyzed.

**Results:** Among 192 participants, 81 were diagnosed with MetS. Both fasting and postprandial ApoB48 concentrations were higher in the MetS group than in the control group ( $P < 0.05$ ). The incidence of MetS increased with rising ApoB48 levels. In both groups, ApoB48 concentrations initially increased and then decreased after OFTT, peaking at 4 h postprandially, with higher peak values observed in the MetS group. Fasting and 2-h postprandial ApoB48 levels had the strongest correlations with MetS.

**Conclusion:** Higher ApoB48 levels before and after OFTT were positively correlated with an increased risk of MetS and were higher than those in the healthy population. Fasting ApoB48 and 2-h postprandial ApoB48 levels after a high-fat meal may be potential markers of MetS.

**Keywords:** high-fat diet, oral fat tolerance testing, intestinal chylomicrons, triglycerides

## Introduction

Metabolic syndrome (MetS) is a group of clinical syndromes mainly characterized by obesity, hyperglycemia (impaired glucose regulation or diabetes), hypertension, and dyslipidemia. These disorders are intrinsically connected. The prevalence of MetS is increasing annually, and approximately 34.7% of American adults suffer from MetS.<sup>1</sup> Globally, as of 2020, approximately 60 million children and adolescents are affected.<sup>2</sup> MetS is associated with various diseases,<sup>3-5</sup> especially cardiovascular disease (CVD), and is a typical risk factor for its development.<sup>6</sup> It increases the relative risk of CVD by 1.53–2.18%, the all-cause mortality rate by 1.27–1.60%, and the risk of type 2 diabetes mellitus (T2DM) by fivefold.<sup>7</sup> It also reduces the quality of life of patients and, in severe cases, may significantly shorten life expectancy.<sup>8</sup> Therefore, it is important to identify relevant risk factors for MetS.

Apolipoproteins are important factors associated with metabolic disorders. Our previous research demonstrated the role of apolipoprotein CIII (ApoCIII) in lipid metabolism and emphasized its association with MetS.<sup>9</sup> Apolipoprotein B (ApoB) is a common apolipoprotein, and apolipoprotein B-48 (ApoB48), a subtype of the ApoB family, is synthesized in the intestine. It participates in CM assembly and secretion and transports triglycerides (TG) from food sources into the blood. When these lipoproteins and residual lipoprotein particles (rlp) accumulate, it can lead to insulin resistance, metabolic disorders and abnormal lipids, increasing the risk of metabolic diseases.<sup>10-12</sup>

In recent years, an increasing number of studies have begun to focus on the association between ApoB48 levels and MetS. Multiple cross-sectional studies have shown that compared with healthy individuals, patients with MetS exhibit significantly elevated ApoB48 levels, and these levels are positively correlated with hypertriglyceridemia and insulin resistance.<sup>13–15</sup> Wu et al discovered that in obese individuals, fasting ApoB48 was approximately 50% higher, while in non-fasting states, the level of ApoB48 was even twice that of people with healthy weight, and it was positively correlated with TG. They also found that an increase of 1mg/mL in APOB48 predicted that the thickness of carotid artery plaques might increase by 0.75mm.<sup>16</sup> However, the existing research still has some limitations. Firstly, the sample size of most studies is limited. Secondly, most articles only observed the fasting state. However, in the fasting state, the concentration of ApoB48 in the blood circulation is usually low, which may make the test results less sensitive. Moreover, people spend most of their day in the postprandial state. Therefore, the changes in lipid metabolism after meals cannot be ignored. To address this issue, OFTT can be used as a functional detection method. By consuming high-fat meals, it dynamically simulates postprandial lipid metabolism changes, thereby conducting association analysis between postprandial ApoB48 dynamics (such as peak time). Thirdly, the current research is still insufficient in analyzing specific components (such as hypertension, etc).

Therefore, this study aimed to analyze the metabolic changes in ApoB48 under fasting and postprandial conditions using a high-fat meal test in a larger sample cohort, and quantify its correlation with each component of MetS, with the aim of providing more accurate experimental evidence for the early identification and risk stratification of MetS.

## Materials and Methods

### Participants

From January to June 2020, volunteers were randomly recruited from the Endocrinology Department of Hebei General Hospital. This study adhered to the principles of the Declaration of Helsinki and was approved by Hebei General Hospital (Registration Number: hiCTR1800019514. Ethical approval number: 2020-KY-27). All participants provided written informed consent.

### Exclusion Criteria

Exclusion criteria were as follows: malignant tumors, vegetarians, heart disease, digestive system diseases, thyroid dysfunction, history of severe diabetes, infectious diseases, kidney diseases, blood system diseases, mental disorders, pregnancy, and use of drugs related to lipid metabolism (such as hormones, beta-blockers, contraceptives, or diuretics). Participants with a history of stroke, trauma, surgery, weight change of more than 3 kg, or severe infection within the past 3 months were also excluded.

All participants underwent an oral glucose tolerance test (OGTT) before the trial. According to the 1999 WHO diagnostic criteria for T2DM (fasting blood glucose  $\geq 7.0$  mmol/L and/or OGTT 2-h blood glucose  $\geq 11.1$  mmol/L), those with abnormal blood glucose were excluded.

### Oral Fat Tolerance Testing (OFTT)

Participants were required to follow a normal diet for one week before oral fat tolerance testing (OFTT). They fasted from 10 pm the night before the OFTT. Fasting blood samples were collected at 8 am the following day. Each participant consumed a high-fat meal containing 1500 kcal of calories (20% from protein, 20% from carbohydrates, and 60% from fat) within 10 min. The levels of total cholesterol (TC), TG, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and ApoB48 were measured 2, 4, 6, and 8 h postprandially. Food intake was prohibited during the test; however, drinking water was allowed.<sup>17</sup>

### Collection of General Characteristics and Detection of Biochemical Indicators

General features included weight, height, and waist circumference (WC). Body mass index (BMI) was calculated as weight (kg) divided by height squared ( $m^2$ ). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured automatically using an electronic blood pressure monitor. After three consecutive automatic repeated measurements, the

average value was considered the participant's blood pressure. Fasting concentrations of TC, TG, HDL-C, LDL-C, ApoB48, and fasting blood glucose (FBG) were determined using the Hitachi 7600 fully automatic biochemical analyzer (Hitachi Instruments Ltd., Tokyo, Japan). Triglyceride-rich lipoprotein residue (TRLR) and non-high-density lipoprotein cholesterol (non-HDL-C) were calculated using the following formulas:  $\text{TRLR} = \text{TC} - \text{HDL-C} - \text{LDL-C}$  and  $\text{non-HDL-C} = \text{TC} - \text{HDL-C}$ , respectively. We also calculated the area under the ApoB48 curve ( $\text{AUC}_{\text{ApoB48}}$ ) within 8 h using the trapezoidal method. Fasting insulin (FINS) concentration and 2-h postprandial insulin (2-h PINS) levels were determined using chemiluminescence. The homeostasis model assessment of insulin resistance (HOMA-IR) was performed using fasting insulin (mIU/L) and fasting glucose (mmol/L):  $\text{HOMA-IR} = \text{FBG (mmol/L)} \times \text{FINS (mIU/L)} / 22.5$ .<sup>18</sup>

## Diagnostic Criteria and Grouping

Based on the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III), the diagnostic criteria for MetS are three or more of the following: (1) abdominal obesity: WC  $\geq 102$  cm for men, WC  $\geq 88$  cm for women; (2) fasting TG  $\geq 1.7$  mmol/L (150 mg/dL) or receiving TG-lowering treatment; (3) fasting HDL-C: male  $< 1.0$  mmol/L (40 mg/dL), female  $< 1.3$  mmol/L (50 mg/dL), or receiving treatment for elevated HDL-C; (4) blood pressure: SBP  $\geq 130$  mmHg or DBP  $\geq 85$  mmHg or receiving antihypertensive treatment; (5) FBG:  $\geq 5.6$  mmol/L (100 mg/dL) or receiving hypoglycemic treatment.<sup>19</sup> Participants were divided into two groups: MetS and control.

## Statistical Analysis

The Shapiro–Wilk method is used to determine the normality of data. Data following a normal distribution were expressed as mean  $\pm$  standard deviation; otherwise, the interquartile range was used. Data from both groups were compared using *t*-tests for normal distribution and non-parametric test (Mann–Whitney *U*-test) for skewed distribution. Pearson correlation analysis was used for the correlation analysis of the two variables if they conformed to a normal distribution and had uniform variances; otherwise, Spearman's analysis was used. A correlation study between MetS and its risk factors was conducted using univariate and multivariate binary logistic regression analyses. The power calculation was carried out using the software G.power.3.1.9.7 (Effect size at 0.5.  $\alpha$ err prob = 0.05. Two tails. The power (1 -  $\beta$ err prob) = 0.8). All figures were generated using GraphPad Prism 8.0 (version 9.0, San Diego, CA, USA). Statistical analyses were conducted using IBM SPSS Statistics for Windows (version 26.0; IBM Corp., Armonk, NY, USA). Statistical significance was set at  $P < 0.05$ .

## Results

### Comparison of General Information Between the Two Groups

Clinical data were collected from 192 volunteers, including 97 women and 95 men, aged 22–67 years, with BMI values ranging from 18.2 kg/m<sup>2</sup> to 35.2 kg/m<sup>2</sup>. Some patients were overweight. The mean fasting ApoB48 level for all volunteers was  $5.14 \pm 1.65$  mg/L (Table 1).

Participants were divided into two groups according to the NCEP ATP III diagnostic criteria. Compared with the control group, the MetS group had significantly higher values for age, SBP, DBP, BMI, WC, TG, TC, LDL-C, TRLR, non-HDL-C, ApoB48, FBG, 2-h postprandial blood glucose (2-h PBG), FINS, 2-h PINS, and HOMA-IR ( $P < 0.05$ ), while HDL-C levels were lower ( $P < 0.05$ ) (Table 1).

We further compared differences according to sex. The control group comprised 49 men and 62 women, while the MetS group comprised 46 men and 35 women. Among men, height, SBP, BMI, WC, TG, TC, LDL-C, TRLR, non-HDL-C, ApoB48, FBG, 2-h PBG, FINS, and HOMA-IR were all higher in the MetS group than in the control group ( $P < 0.05$ ). Among women, age, weight, SBP, DBP, BMI, WC, TG, TC, LDL-C, TRLR, non-HDL-C, ApoB48, FBG, 2-h PBG, FINS, 2-h PINS, and HOMA-IR were higher in the MetS group than in the control group ( $P < 0.05$ ). In both sexes, HDL-C levels were lower in the MetS group than in the control group ( $P < 0.05$ ) (Table 2).

**Table 1** Anthropometric and Biochemical Characteristics of All Chinese Study Subjects

	Total (N=192)	Con (N=111)	MetS (N=81)	P
Gender (Men/women)	95/97	49/62	46/35	
Age (years)	43.82±12.36	41.30±12.77	47.01±10.87	0.003
Height (cm)	165.74±7.46	166.20±8.09	164.58±6.28	0.234
Weight (kg)	69.72±10.86	69.15±11.34	70.77±10.33	0.181
SBP (mmHg)	123.42±12.69	121.23±12.06	126.99±12.81	0.001
DBP (mmHg)	77.69±7.68	76.66±7.54	79.51±7.70	0.008
BMI (kg/m <sup>2</sup> )	25.46±3.28	24.98±3.34	26.07±3.17	0.013
WC (cm)	87.58±10.42	83.91±10.44	92.54±8.25	<0.001
TG (mmol/L)	1.41±0.57	1.13±0.36	1.79±0.53	<0.001
TC (mmol/L)	4.63±0.92	4.48±0.82	4.82±1.02	0.011
HDL-C (mmol/L)	1.27±0.34	1.37±0.35	1.13±0.28	<0.001
LDL-C (mmol/L)	2.95±0.71	2.78±0.61	3.11±0.71	0.009
TRLR (mmol/L)	0.43 (0.33, 0.57)	0.40 (0.27, 0.50)	0.52 (0.38, 0.71)	<0.001
Non-HDL-C (mmol/L)	3.39±0.86	3.20±0.74	3.69±0.92	<0.001
ApoB48 (mg/L)	5.14±1.65	4.77±1.64	5.65±1.52	<0.001
FBG (mmol/L)	5.28 (4.92, 5.68)	5.06 (4.83, 5.53)	5.66 (5.20, 6.01)	<0.001
2hPBG (mmol/L)	5.90 (4.95, 6.90)	5.58 (4.80, 6.54)	6.13 (5.32, 7.53)	0.001
FINS (mU/L)	10.25 (7.82, 13.96)	9.80 (6.90, 12.18)	11.53 (9.15, 17.69)	<0.001
2hPINS (mU/L)	68.82 (39.09, 106.83)	56.80 (33.95, 88.92)	84.65 (51.89, 126.00)	<0.001
HOMA-IR	2.47 (1.78, 3.30)	2.15 (1.58, 2.97)	2.78 (2.29, 4.30)	<0.001

**Notes:** Means ± SD for normally distributed variables, M50 (M25, M75) for non-normally distributed variables.

**Abbreviations:** SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; WC, waist circumference; TG, triglyceride; TC, total cholesterol; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; TRLR, triglyceride-rich lipoproteins residue; ApoB48, apolipoprotein B48; FBG, fasting blood glucose; 2hPBG, blood glucose 2h postprandially; FINS, fasting plasma insulin concentration; 2hPINS, plasma insulin concentration 2h postprandially; HOMA-IR, homeostasis model assessment of insulin resistance.

**Table 2** Comparison of Anthropometric, Clinical and Fasting Metabolic Parameters in Participants of Each Sex

	Con Women	Men	MetS Women	Men
Age (years)	41.87±13.05	41.18±12.95	48.26±9.45 <sup>a</sup>	45.87±11.77
Height (cm)	160.73±5.68	172.15±4.86	159.21±4.24	168.95±8.09 <sup>b</sup>
Weight (kg)	61.38±7.66	75.04±9.21	69.17±8.84 <sup>a</sup>	76.35±8.81
SBP (mmHg)	116.39±9.79	124.88±11.43	124.97±13.02 <sup>a</sup>	130.17±12.93 <sup>b</sup>
DBP (mmHg)	74.71±6.60	78.08±7.88	78.14±8.42 <sup>a</sup>	80.96±6.93
BMI (kg/m <sup>2</sup> )	23.79±3.00	25.29±2.72	27.28±3.26 <sup>a</sup>	26.73±2.69 <sup>b</sup>
WC (cm)	79.51±8.01	88.67±10.16	90.16±8.22 <sup>a</sup>	95.32±7.45 <sup>b</sup>
TG (mmol/L)	1.13±0.32	1.07±0.35	1.73±0.51 <sup>a</sup>	1.91±0.58 <sup>b</sup>
TC (mmol/L)	4.60±0.87	4.33±0.69	4.93±1.19 <sup>a</sup>	4.74±0.89 <sup>b</sup>
HDL (mmol/L)	1.42±0.31	1.32±0.37	1.15±0.22 <sup>a</sup>	1.12±0.33 <sup>b</sup>
LDL-C (mmol/L)	2.90±0.78	2.74±0.53	3.18±0.82 <sup>a</sup>	3.08±0.64 <sup>b</sup>
TRLR (mmol/L)	0.34 (0.19, 0.49)	0.35 (0.13, 0.49)	0.48 (0.38, 0.71) <sup>a</sup>	0.55 (0.35, 0.76) <sup>b</sup>
Non-HDL-C (mmol/L)	3.18±0.82	3.01±0.78	3.78±1.14 <sup>a</sup>	3.63±0.88 <sup>b</sup>
ApoB48 (mg/L)	4.97±1.76	4.51±1.46	5.70±1.18 <sup>a</sup>	5.61±1.74 <sup>b</sup>
FBG (mmol/L)	5.02 (4.82, 5.26)	5.09 (4.78, 5.56)	5.64 (4.98, 5.89) <sup>a</sup>	5.67 (5.31, 6.11) <sup>b</sup>
2hPBG (mmol/L)	5.60 (4.78, 6.35)	5.30 (4.80, 6.64)	6.25 (5.71, 7.40) <sup>a</sup>	6.13 (5.257.95) <sup>b</sup>
FINS (mU/L)	9.67 (6.46, 12.41)	10.03 (7.50, 12.06)	12.17 (10.36, 17.70) <sup>a</sup>	10.31 (8.48, 17.79) <sup>b</sup>
2hPINS (mU/L)	51.37 (31.47, 84.23)	58.84 (34.86, 91.34)	97.15 (56.06, 126.80) <sup>a</sup>	82.08 (51.43, 122.55) <sup>b</sup>
HOMA-IR	2.15 (1.36, 2.97)	2.23 (1.73, 2.90)	3.09 (2.38, 4.39) <sup>a</sup>	2.79 (2.29, 4.38) <sup>b</sup>

**Notes:** Means ± SD for normally distributed variables, M50 (M25, M75) for non-normally distributed variables. <sup>a</sup>P < 0.05 versus Women in the Con group, <sup>b</sup>P < 0.05 versus Men in the Con group.

**Abbreviations:** SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; WC, waist circumference; TG, triglyceride; TC, total cholesterol; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; TRLR, triglyceride-rich lipoprotein residue; ApoB48, apolipoprotein B48; FBG, fasting blood glucose; 2hPBG, blood glucose 2h postprandially; FINS, fasting plasma insulin concentration; 2hPINS, plasma insulin concentration 2h postprandially; HOMA-IR, homeostasis model assessment of insulin resistance.

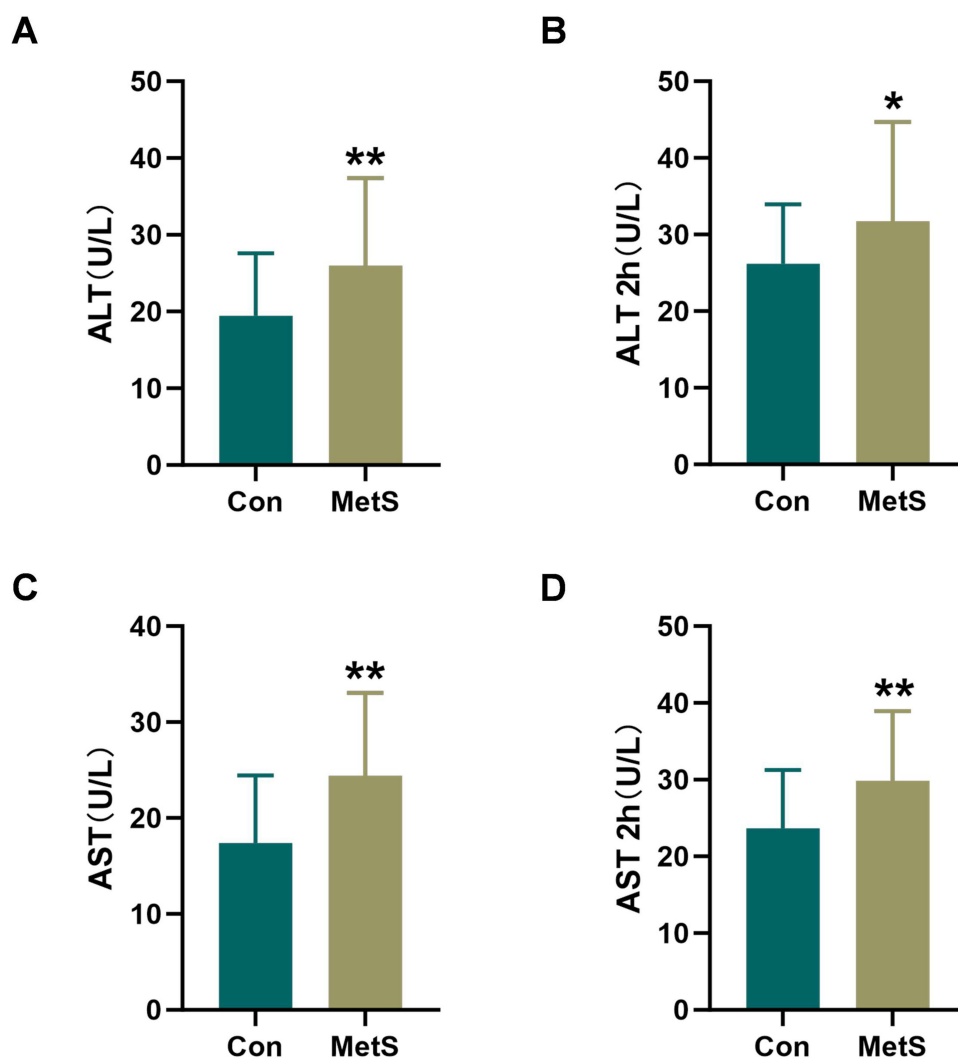
## Comparison of Hepatic Function

This study compared the liver enzymatic indicators of the two groups of subjects. The levels of ALT and AST in the MetS group were significantly higher than those in the control group both fasting and postprandial, and the difference was statistically significant ( $P < 0.05$ ) (Figure 1). It is suggested that patients with metstd have more significant hepatocyte inflammation or metabolic liver damage.

## Comparison of Blood Lipids After OFTT

OFTT was conducted on all participants. At each time point, TG, TC, LDL-C, non-HDL-C, and TRLR levels were higher in the MetS group than in the control group ( $P < 0.05$ ), while HDL-C levels were lower ( $P < 0.05$ ) (Table 3).

There were differences in lipoprotein levels between the two groups before and after a high-fat meal. ApoB48 levels first increased and then decreased in both groups. At each time point, ApoB48 levels in the MetS group were higher than those in the control group ( $P < 0.05$ ), significantly increasing at 2 h and peaking at 4 h with a concentration 1.14 times that of the control group (Figure 2A). Compared with the baseline level (0 h) in the same group, ApoB48 concentrations at 2, 4, 6, and 8 h were significantly increased ( $P < 0.05$ ). The area under the curve (AUC) for ApoB48 over 8 h was also significantly higher in the MetS group than in the control group ( $P < 0.05$ ) (Figure 2B).



**Figure 1** Comparison of the hepatic function. (A) The level of fasting ALT. (B) The ALT level 2 hours after a high-fat meal. (C) The level of fasting AST. (D) The AST level 2 hours after a high-fat meal.

**Notes:** \* $P < 0.05$  versus Con group, \*\* $P < 0.001$  versus Con group.

**Table 3** Changes of Lipids Factors During OFTT in Two Groups

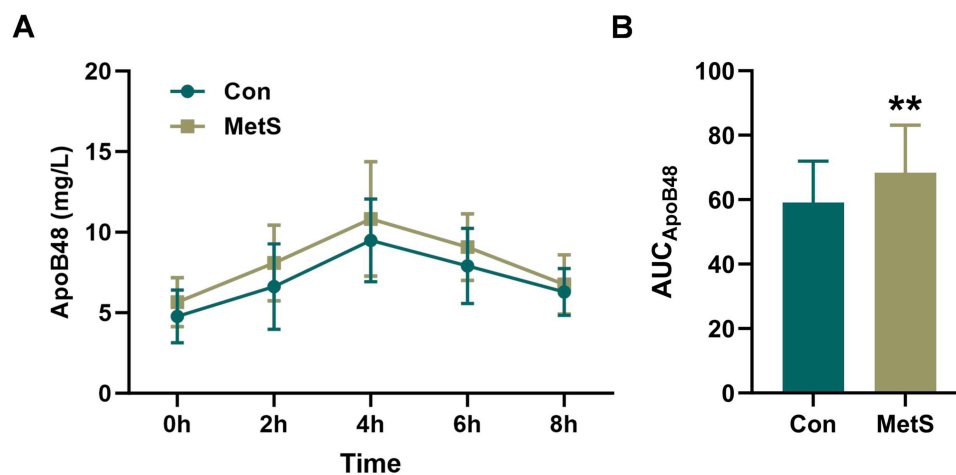
	Group	0h	2h	4h	6h	8h
<b>TG</b>	Con	1.13±0.36	1.92±0.64 <sup>a</sup>	2.26±0.93 <sup>a</sup>	2.09±1.00 <sup>a</sup>	1.78±0.97 <sup>a</sup>
	MetS	1.80±0.57	2.54±0.75 <sup>b</sup>	3.52±1.28 <sup>b</sup>	3.37±1.34 <sup>b</sup>	3.25±1.52 <sup>b</sup>
	P	<0.001	<0.001	<0.001	<0.001	<0.001
<b>TC</b>	Con	4.48±0.80	4.53±0.88	4.50±0.89	4.66±0.96 <sup>a</sup>	4.70±0.97 <sup>a</sup>
	MetS	4.82±1.03	4.82±0.94	4.86±0.98	5.08±1.05 <sup>b</sup>	5.11±1.06 <sup>b</sup>
	P	0.011	0.030	0.009	0.005	0.006
<b>HDL-C</b>	Con	1.37±0.35	1.28±0.25 <sup>a</sup>	1.22±0.23 <sup>a</sup>	1.22±0.24 <sup>a</sup>	1.27±0.24 <sup>a</sup>
	MetS	1.14±0.28	1.17±0.25	1.11±0.26	1.11±0.26	1.15±0.25
	P	<0.001	0.002	0.003	0.002	0.001
<b>LDL-C</b>	Con	2.84±0.68	2.78±0.65 <sup>a</sup>	2.73±0.62 <sup>a</sup>	2.81±0.65 <sup>a</sup>	2.85±0.66 <sup>a</sup>
	MetS	3.11±0.73	3.03±0.69 <sup>b</sup>	2.96±0.67 <sup>b</sup>	3.04±0.70 <sup>b</sup>	3.09±0.71 <sup>b</sup>
	P	0.009	0.012	0.017	0.019	0.020
<b>Non-HDL-C</b>	Con	3.18±0.73	3.24±0.78	3.28±0.81 <sup>a</sup>	3.44±0.88 <sup>a</sup>	3.43±0.88 <sup>a</sup>
	MetS	3.68±0.94	3.64±0.91	3.75±0.94	3.97±1.03 <sup>b</sup>	3.96±1.04 <sup>b</sup>
	P	<0.001	0.001	<0.001	<0.001	<0.001
<b>TRLR</b>	Con	0.40 (0.27, 0.50)	0.42 (0.31, 0.56)	0.52 (0.37, 0.64) <sup>a</sup>	0.56 (0.41, 0.73) <sup>a</sup>	0.51 (0.39, 0.67) <sup>a</sup>
	MetS	0.51 (0.38, 0.71)	0.54 (0.43, 0.75)	0.75 (0.59, 0.94) <sup>b</sup>	0.83 (0.62, 1.03) <sup>b</sup>	0.73 (0.54, 1.04) <sup>b</sup>
	P	<0.001	<0.001	<0.001	<0.001	<0.001
<b>ApoB48</b>	Con	4.77±1.64	6.63±2.66 <sup>a</sup>	9.49±2.56 <sup>a</sup>	7.91±2.34 <sup>a</sup>	6.29±1.46 <sup>a</sup>
	MetS	5.65±1.52	8.09±2.36 <sup>b</sup>	10.83±3.55 <sup>b</sup>	9.07±2.07 <sup>b</sup>	6.76±1.83 <sup>b</sup>
	P	<0.001	<0.001	0.003	<0.001	0.048

**Notes:** Means ± SD for normally distributed variables, M50 (M25, M75) for non-normally distributed variables. <sup>a</sup>P < 0.05 versus 0h in the Con group, <sup>b</sup>P < 0.05 versus 0h in the MetS group.

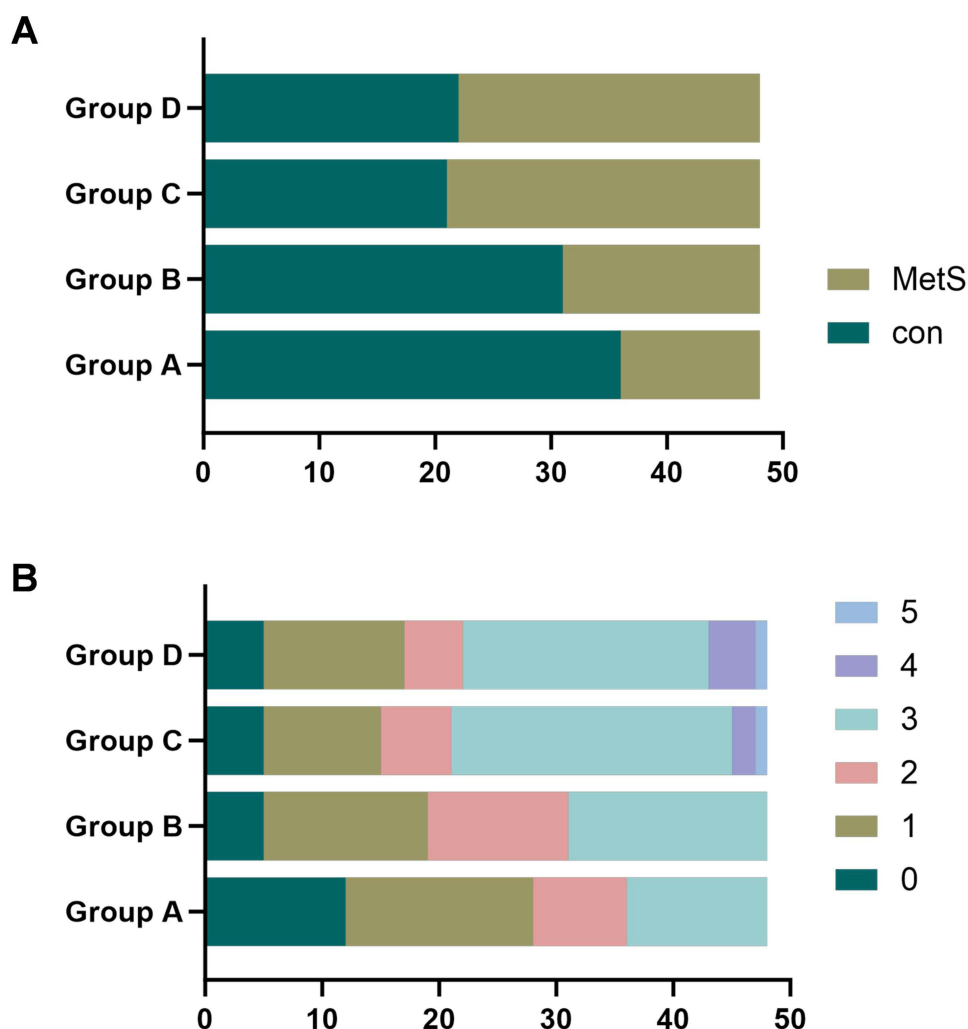
**Abbreviations:** TG, triglyceride; TC, total cholesterol; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; TRLR, triglyceride-rich lipoprotein residue; ApoB48, apolipoprotein B48.

## Trend in MetS Prevalence Based on ApoB48 Levels

Participants were divided into four groups from lowest to highest, based on the interquartile range of fasting ApoB48 levels: Group A (ApoB48 < 3.8500 mg/L), Group B (3.8500 mg/L ≤ ApoB48 < 5.2700 mg/L), Group C (5.2700 mg/L ≤ ApoB48 < 6.3275 mg/L), and Group D (ApoB48 ≥ 6.3275 mg/L). The numbers of patients without and with MetS in each group were as follows: Group A (36 and 12), Group B (32 and 16), Group C (21 and 27), and Group D (22 and 26). The incidence of MetS has significantly increased, ranging from 25–35% to 50–60%. When the number of MetS components (according to the MetS



**Figure 2** Comparison of the ApoB48 after OFTT. **(A)** ApoB48 concentrations at different time points during OFTT in two groups. **(B)** The area under the curve. **Notes:** \*\*P < 0.001 versus Con group.



**Figure 3** Comparison of the number of MetS in the subgroups. **(A)** The number of MetS in each subgroup. **(B)** The number of components of MetS in each subgroup. The numbers represent the number of components containing the diagnostic criteria for MetS.

diagnostic criteria) was reassessed, it was found that higher ApoB48 levels were associated with a higher number of components. Therefore, the incidence of MetS tended to increase with increasing ApoB48 levels (Figure 3).

### Correlation Analysis of ApoB48 and MetS Components Before and After OFTT

ApoB48 concentrations at fasting and 2, 4, 6, and 8 h after OFTT were correlated with each MetS component. Fasting ApoB48 was positively correlated with DBP, WC, FBG, and TG ( $P < 0.05$ ) and negatively correlated with HDL-C, with the strongest correlation observed for HDL-C ( $r = -0.290$ ,  $P < 0.001$ ). 2-h ApoB48 was positively correlated with SBP, DBP, WC, FBG, and TG ( $P < 0.05$ ) and negatively correlated with HDL-C ( $r = -0.261$ ,  $P < 0.001$ ). 4-h ApoB48 was positively correlated with WC and FBG levels ( $P < 0.05$ ) and negatively correlated with HDL-C levels ( $r = -0.241$ ,  $P = 0.001$ ). 6-h ApoB48 was positively correlated with WC, FBG, and TG levels ( $P < 0.05$ ). 8-h ApoB48 was positively correlated with FBG ( $r = -0.151$ ,  $P < 0.05$ ). Overall, ApoB48 most strongly correlated with MetS at 0 and 2 h after a high-fat meal (Table 4).

### Binary Logistic Regression Analysis of MetS

Using MetS as the dependent variable, binary logistic regression was used to analyze the relationship between ApoB48 and MetS. Independent variables were screened successively, revealing that independent variables related to MetS were age, SBP, DBP, BMI, TC, TG, HDL-C, LDL-C, 0-h ApoB48, 2-h ApoB48, 4-h ApoB48, 6-h ApoB48, and HOMA-IR.

**Table 4** Relationship Between Fasting and Postprandial ApoB48 with MetS Components

	0h	2h	4h	6h	8h
SBP	0.093 (0.200)	0.197 (0.006)	0.123 (0.088)	0.132 (0.069)	0.017 (0.815)
DBP	0.143 (0.048)	0.186 (0.010)	0.135 (0.062)	0.126 (0.083)	0.039 (0.596)
WC	0.210 (0.004)	0.233 (0.001)	0.240 (0.001)	0.247 (0.001)	0.062 (0.391)
FBG	0.197 (0.006)	0.221 (0.002)	0.204 (0.005)	0.211 (0.003)	0.151 (0.036)
HDL-C	-0.290 (<0.001)	-0.261 (<0.001)	-0.241 (0.001)	-0.097 (0.180)	0.020 (0.786)
TG	0.209 (0.004)	0.184 (0.011)	0.068 (0.352)	0.193 (0.007)	0.030 (0.683)

**Notes:** Data for r (P). r, correlation coefficient.

**Abbreviations:** SBP, systolic blood pressure; DBP, diastolic blood pressure; WC, waist circumference; FBG, fasting blood glucose; HDL-C, high-density lipoprotein-cholesterol; TG, triglyceride.

The relationship between ApoB48 levels and MetS was analyzed after adjusting for age and sex. We found that 0-h, 2-h, 4-h, and 6-h ApoB48 levels were positively correlated with MetS. Candidate variables ( $P < 0.01$ ), such as SBP, TG, HDL-C, and HOMA-IR, were included in the multivariate binary logistic regression equation. After readjustment, fasting and 2-h ApoB48 remained independent risk factors for MetS (odds ratio [OR] = 1.335,  $P = 0.042$ ; OR = 1.196;  $P = 0.045$ , respectively). For each unit increase in fasting ApoB48 levels, the probability of developing MetS increased by 33.5%, and for each unit increase in 2-h ApoB48 levels, the probability of developing MetS increased by 19.6% (Table 5).

**Table 5** Binary Logistic Regression Analysis to Assess the MetS

		B	SE	OR (95% CI)	Wald	P
<b>Model 1</b>	Age	0.036	0.012	1.037 (1.012, 1.063)	8.497	0.004
	Gender	0.336	0.293	1.399 (0.787, 2.486)	1.311	0.252
	SBP	0.042	0.012	1.043 (1.017, 1.068)	11.165	0.001
	DBP	0.052	0.020	1.053 (1.013, 1.095)	6.664	0.010
	BMI	0.114	0.047	1.121 (1.022, 1.229)	5.916	0.015
	TC	0.414	0.165	1.513 (1.094, 2.093)	6.271	0.012
	TG	3.095	0.449	22.094 (9.161, 53.288)	47.486	<0.001
	HDL-C	-2.592	0.581	0.075 (0.024, 0.234)	19.903	<0.001
	LDL-C	0.547	0.214	1.728 (1.135, 2.631)	6.506	0.011
	ApoB48	0.348	0.098	1.416 (1.169, 1.715)	12.693	<0.001
	2h ApoB48	0.225	0.061	1.253 (1.111, 1.413)	13.499	<0.001
	4h ApoB48	0.146	0.050	1.157 (1.049, 1.278)	8.416	0.004
	6h ApoB48	0.238	0.072	1.271 (1.102, 1.464)	10.925	0.001
8h ApoB48	0.180	0.092	1.197 (1.000, 1.434)	3.822	0.051	
HOMA-IR	0.505	0.125	1.657 (1.297, 2.116)	16.342	<0.001	
<b>Model 2</b>	ApoB48	0.422	0.105	1.525 (1.241, 1.874)	16.149	<0.001
	2h ApoB48	0.236	0.063	1.266 (1.119, 1.433)	13.951	<0.001
	4h ApoB48	0.149	0.052	1.161 (1.049, 1.284)	8.307	0.004
	6h ApoB48	0.232	0.073	1.262 (1.093, 1.456)	10.086	0.001
	8h ApoB48	0.160	0.094	1.173 (0.977, 1.410)	2.913	0.088
<b>Model 3</b>	ApoB48	0.289	0.142	1.335 (1.010, 1.764)	4.120	0.042
	2h ApoB48	0.179	0.089	1.196 (1.004, 1.424)	4.018	0.045
	4h ApoB48	0.106	0.074	1.112 (0.962, 1.285)	2.076	0.150
	6h ApoB48	0.180	0.097	1.198 (0.990, 1.448)	3.465	0.063
	8h ApoB48	0.243	0.134	1.274 (0.980, 1.658)	3.268	0.071

**Notes:** Model 1: without adjustment, crude OR. Model 2: Based on Model 1, further adjusted for sex age.

Model 3: Based on Model 2, further adjusted for SBP, TG, HDL-C, HOMA-IR.

**Abbreviations:** SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; ApoB48, apolipoprotein B48; 95% CI, 95% confidence interval.

## Discussion

ApoB48 is an apolipoprotein component of CM and is present in CM-Rs derived from CM fat breakdown.<sup>20</sup> Each lipoprotein particle of CM and CM-R contains one ApoB48 molecule; therefore, ApoB48 levels reflect the quantity of ApoB48-containing lipoproteins. Direct measurement of serum ApoB48 can more accurately assess lipoprotein content.

Although the role of LDL-C in metabolic diseases is well known,<sup>21</sup> the “ApoB principle” has been proposed in recent years. Multiple studies have shown that ApoB100 and ApoB48 are involved in the development of metabolic diseases such as diabetes,<sup>22</sup> atherosclerotic cardiovascular disease,<sup>23–26</sup> and non-alcoholic fatty liver disease (NAFLD).<sup>17</sup> Therefore, residual lipoproteins may be key factors leading to metabolic diseases. Our previous research found that ApoB concentrations vary among populations and are correlated with glycolipid metabolism.<sup>27,28</sup> In this study, participants were divided into MetS and control groups to compare differences in ApoB48, with the aim of exploring the relationship between ApoB48 and MetS. To date, only a few studies have been conducted on ApoB48 and MetS,<sup>13</sup> focusing on the concentration of fasting ApoB48. Because fasting levels mainly refer to liver-derived lipoproteins, due to the delayed decomposition of liver fat,<sup>29</sup> and may not promptly reflect current abnormalities, hidden metabolic dysfunctions may be missed. Moreover, individuals spend most of the day in a postprandial state,<sup>24</sup> during which residual lipoproteins continuously produced after each meal act as additional risk factors. Therefore, changes in lipoprotein levels after meals should also be analyzed.<sup>30,31</sup> Postprandial hypertriglyceridemia (PHTG), characterized by the postprandial accumulation of enterogenous CM and CM-R, is an independent risk factor for metabolic diseases.<sup>32</sup>

Recently, a proteomic analysis study confirmed that CM-Rs have atherosclerotic characteristics through ultracentrifugation and immunoprecipitation using an ApoB48-specific antibody.<sup>14</sup> Similarly, ApoB48 was detected in atherosclerotic plaques in a clinical study.<sup>33</sup> Therefore, ApoB48 levels are strongly correlated with MetS. Jin et al showed that fasting ApoB48 levels may help predict CVD.<sup>34</sup> During the follow-up period of a clinical study, Szili-Torok et al found that cardiovascular events and total mortality were significantly associated with baseline ApoB48 levels (hazard ratio [HR] = 1.05, OR = 1.13).<sup>35</sup> In addition, several studies have investigated postprandial ApoB48. Lim et al found that patients with T2DM showed an increase in ApoB48 AUC after a high-fat meal.<sup>36</sup> Riitta et al reported similar findings. By analyzing 11 patients with diabetes who consumed a fat-rich diet containing 927 kcal, they found no significant difference in fasting lipid profiles, whereas postprandial ApoB48 and residue levels increased twofold.<sup>15</sup> These differences are mainly attributed to the excessive intestinal secretion of ApoB48-containing lipoproteins into CMs and the low fat decomposition rate of CMs. Overall, previous studies have demonstrated that both fasting and postprandial ApoB48 levels have a certain degree of predictive value when assessing the risk of MetS and cardiovascular and cerebrovascular diseases.<sup>37</sup> In the current study, ApoB48 levels were higher in the MetS group than in the control group both before and after a high-fat meal. Therefore, changes in ApoB48 levels are crucial risk markers for MetS.

In this study, the basic characteristics of the two groups were first analyzed, including BMI, WC, TG, TC, LDL-C, and HOMA-IR, all of which were significantly higher in the MetS group than in the control group. The 2022 report from the US Centers for Disease Control and Prevention also indicated that obese adults were 4.74 times more likely to develop MetS,<sup>3</sup> consistent with a previous study.<sup>38</sup> We further found positive correlations among BMI, WC, and MetS. Considering potential sex differences in lipid metabolism, baseline values were compared within each sex group. MetS participants had higher values than the sex-matched control group. However, contrary to previous research conclusions,<sup>13,39</sup> we found no significant sex difference in fasting ApoB48 concentration. This discrepancy may be affected by racial differences, as previous studies were based on Australian youths and Japanese adults, while this study focused solely on local Chinese adults, resulting in certain differences in measured values. We also found both ALT and AST are liver-specific indicators. Their elevation usually directly reflects the degree of steatosis and inflammation of liver cells, that is, non-alcoholic fatty liver disease (NAFLD), and NAFLD is closely related to insulin resistance and MetS. The steatosis environment of the liver may have an intrinsic connection with the clearance disorder of chylomicrons from the intestine (rich in ApoB48), which provides new and direct evidence for the study of the mechanism.

We observed that ApoB48 concentration peaked 4 h after consumption of a high-fat meal containing 100 g of fat. Taskinen et al reached the same conclusion when they fed participants a high-fat meal containing 68 g of fat to study the

secretion and clearance of TG lipoproteins.<sup>15</sup> Although there were slight differences in fat load, the changing trends in ApoB48 were similar.

Correlation analysis showed that fasting and 2-h postprandial ApoB48 were significantly associated with all MetS components, whereas 4-h and 6-h ApoB48 levels were associated with two MetS components, and 8-h ApoB48 with only one component. These associations were verified using multivariate logistic regression analysis. Similar to previous studies,<sup>40</sup> we also found that insulin resistance is a central link in MetS. However, fasting and 2-h ApoB48 levels after a high-fat meal remained correlated with MetS after adjusting for sex, age, SBP, TG, HDL-C, and HOMA-IR, supporting the reliability of our findings. In addition, this study revealed that as the concentration of ApoB48 increased, the incidence of MetS increased gradually.

Currently, no reference range exists for fasting ApoB48 levels in Chinese adults or adolescents. A study involving a large number of adolescents ( $n = 1045$ ) reported an average fasting ApoB48 level of 13.93 mg/L,<sup>13</sup> while a study on healthy adult men in Japan suggested a reference upper limit of 5.7 mg/L,<sup>32</sup> which was different from the fasting baseline value of  $5.14 \pm 1.65$  mg/L in our study. Changes in leptin, adiponectin, and other factors affecting insulin sensitivity at different growth stages may contribute to these different concentrations of ApoB48. Future studies with a larger number of participants will be needed to formulate corresponding reference ranges for different demographic groups.

## Study Strengths and Limitations

This study used the OFTT to observe the changes in ApoB48 levels under fasting and postprandial conditions, which have not been previously reported. Stratified analyses revealed that higher ApoB48 concentrations were associated with increased prevalence of MetS. Correlation and regression analyses also revealed that baseline fasting ApoB48 levels and postprandial 2-h ApoB48 levels were significantly increased in the MetS group, emphasizing the importance of considering intestinal lipoprotein metabolism in MetS.

However, this study has some limitations. First, the sample size is small and limited to a local population, which may affect the generalizability of the results. Second, as a cross-sectional study, causality between ApoB48 and MetS could not be determined. Multicenter prospective studies are needed to confirm our findings and to explore the specific mechanism behind the observed associations. Third, the detection of ApoB48 may be influenced by hyperlipidemia, interactions with other lipoproteins, and detection methods. The roles of several lipoproteins, such as ApoB, ApoB48, and ApoB100, in MetS should be further explored. Fourth, despite adjusting for key demographic and clinical variables (gender, age, blood pressure, HOMA-IR) in the logistic regression analysis model, residual confounding, including those that may be caused by lifestyle and dietary habits, nutritional status, racial differences in the population, socioeconomic status and other factors, cannot be completely ruled out, as we were limited by data availability and the observational nature of our study design. Controlling for these factors may enhance or weaken the observed associations. Future research needs to obtain more comprehensive datasets to explain these important confounding factors. The proportion of high-fat meals may also cause variations in ApoB48 levels. Future exploratory studies should be conducted using standardized fat-rich meals.

Regarding the clinical implications of our findings, the identified association suggests that ApoB48 is a modest, independent risk marker for MetS. This knowledge could be valuable for refining risk stratification models in clinical practice. For example, patients with elevated levels of ApoB48 might be prioritized for more intensive screening or preventive interventions. Furthermore, this association generates a hypothesis for future interventional studies to determine whether modifying ApoB48 can lead to a reduction in the incidence of MetS. It is crucial to emphasize that our results are preliminarily highlight a potential pathway for risk mitigation.

## Conclusion

In summary, we found that fasting ApoB48 and 2-h ApoB48 levels after a high-fat meal were significantly and positively correlated with MetS. These findings suggest that ApoB48 may be a potential marker for MetS, providing a fundamental basis for identifying at-risk patients through residual lipoprotein levels and reducing the prevalence of MetS by reducing intestinal lipid absorption. This could provide relevant guidance for clinical application.

## Abbreviations

MetS, metabolic syndrome; OFTT, oral fat tolerance test; SBP, systolic blood pressure; DBP diastolic blood pressure; BMI, body mass index; WC, waist circumference; TC, total cholesterol; TG, triglyceride; HDL-C, low-density lipoprotein; LDL-C, low-density lipoprotein cholesterol; TRLR, triglyceride-rich lipoprotein residue; non-HDL-C, non-high-density lipoprotein cholesterol; ApoB48, apolipoprotein B 48; FBG, fasting blood glucose; FINS, fasting insulin; HOMA-IR homeostasis model assessment of insulin resistance.

## Data Sharing Statement

The original contributions presented in the study are included in the article. Further inquiries can be directed to the corresponding author.

## Ethics Approval and Informed Consent

The studies involving humans were approved by Hebei General Hospital Ethics Committee. The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study.

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## Author Contributions

YT: Data curation, Methodology, Writing - original draft, Investigation, Validation; SZ, XH and PT: Formal analysis, Conceptualization, Methodology, Supervision; LR: Conceptualization, Project administration, Writing - review & editing; GS: Conceptualization, Methodology, Supervision, Project administration, Writing-Reviewing and Editing. All authors gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agreed to be accountable for all aspects of the work.

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## Disclosure

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest. The authors have no relevant financial or non-financial interests to disclose.

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