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ORIGINAL RESEARCH

Association Between Weight-Adjusted Waist Circumference Index and Metabolic Disease-Associated Fatty Liver Disease: A Retrospective Study

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Background: Metabolic disease-associated fatty liver disease has brought significant challenges to public health and social-economics. There is a need for a straightforward and effective method to screen for metabolic disease-associated fatty liver disease. The weight-adjusted waist circumference index offers a comprehensive reflection of visceral fat accumulation and skeletal muscle loss. This study aims to explore the relationship between weight-adjusted waist circumference index and hepatic steatosis and hepatic fibrosis.

Methods: This single-centered study screened 288 participants from the outpatient department of the Affiliated Hospital of Hangzhou Normal University (Hangzhou, China). Multiple linear regression models were utilized to assess the association between weight-adjusted waist circumference index and hepatic steatosis and hepatic fibrosis. The subgroup analysis was carried out according to sex, smoking, drinking, BMI, cardiovascular disease, diabetes and hypertension.

Results: Metabolic disease-associated fatty liver disease was diagnosed in 185 out of 288 patients. Multivariate linear regression analysis indicated a positive linear correlation between weight-adjusted waist circumference index and hepatic steatosis and hepatic fibrosis ($P < 0.05$). Analysis of subgroups revealed a stronger positive correlation between weight-adjusted waist circumference index and controlled attenuation parameter among participants aged 18–59 years and smokers ($P < 0.05$). Positive correlations between weight-adjusted waist circumference index and hepatic fibrosis were observed in participants who were alcohol consumers, male, had a body mass index of >28, and with diabetes ($P < 0.05$).

Conclusion: We report a significant linear positive correlation between weight-adjusted waist circumference index and metabolic disease-associated fatty liver disease, suggesting that weight-adjusted waist circumference index is a potential indicator for metabolic disease-associated fatty liver disease screening.

Keywords: hepatic steatosis, hepatic fibrosis, abdominal obesity, weight-adjusted waist index, metabolic-associated fatty liver disease, cross-sectional study

Background

Non-alcoholic fatty liver disease (NAFLD) has emerged as the predominant liver disorder worldwide, presenting a significant challenge to public health and socioeconomic systems.[1] In Asia, the prevalence of NAFLD surpasses the global average, reaching 27.37%, and in China, the prevalence of NAFLD has increased from 17% in 2003 to 22.4% in

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2012.[2,3] NAFLD is intricately linked to metabolic abnormalities, especially overweight/obesity and type 2 diabetes mellitus (T2DM),[4] with obesity directly contributing to its rising incidence.[2,5] To better characterize these fatty liver conditions in the context of metabolic dysfunction, an international expert group proposed redefining NAFLD as metabolic-associated fatty liver disease (MAFLD).[6,7] Compared with NAFLD, MAFLD is more adept at identifying individuals at a high risk of fatty liver disease.[8] Importantly, MAFLD increases the risk of various complications, including intrahepatic complications such as cirrhosis and hepatocellular carcinoma, and extrahepatic complications such as cardiovascular disease and extrahepatic malignancies.[2] Hence, accurate assessment of hepatic steatosis and hepatic fibrosis in MAFLD is essential. However, traditional diagnostic methods such as B-ultrasound and CT entail high costs,[9] while the liver biopsy is invasive.[10,11] And vibration-controlled transient elastography suffers from accuracy limitations.[12–14] Consequently, there is an urgent need for an economical and simple MAFLD screening approach to alleviate the burden it imposes on individuals, families, and healthcare systems.

Both increased body mass index (BMI) and waist circumference (WC) contribute to the degree of fibrosis in MAFLD, as evidenced by established correlations.[15] While BMI and WC are common obesity indices, BMI does not distinguish between subcutaneous fat from visceral fat.[16] To better capture abdominal obesity, Park et al introduced a novel obesity anthropometric index—the weight-adjusted waist circumference index (WWI). Calculated as WC divided by the square root of body weight, WWI more accurately reflects centrally distributed obesity independent of weight.[17] Furthermore, compared with BMI and WC, WWI has demonstrated consistent applicability among different racial and ethnic groups and exhibits superior accuracy in predicting cardiovascular disease mortality risk compared to BMI and WC.[18,19] However, the research on the relationship between WWI and MAFLD is controversial. A previous study by Shen et al found that the relationship between WWI and hepatic fibrosis is nonlinear, and the relationship between WWI and hepatic steatosis is linear, A linear correlation between WWI and hepatic fibrosis was found by Hu et al[20,21] Therefore, further investigation is needed to elucidate this relationship.

In order to understand the relationship between obesity and MAFLD more accurately, this study aims to investigate the relationship between WWI and MAFLD using data collected from the Affiliated Hospital of Hangzhou Normal University.

Method

Study Population

This unique cross-sectional study, conducted at a single center on behalf of the Chinese Medical Doctor Association Tasly Fatty Liver College, aimed to investigate the association between WWI and MAFLD. Outpatients were recruited from the Affiliated Hospital of Hangzhou Normal University between March 1, 2021, and October 1, 2023. This study is a retrospective research project that has obtained approval from the Ethics Committee of Hangzhou Normal University Affiliated Hospital and informed consent has been waived (No.2024 (E2) -KS- 024). The study complied with the provisions of the Declaration of Helsinki and protects the personal privacy of research subjects, ensuring data confidentiality.

Initially, 309 non-pregnant participants were recruited. We further excluded participants aged <18 years (n = 2), lacking controlled attenuation parameter (CAP) data (n = 4), lacking WC data (n = 6), lacking weight data (n = 1), and those with duplicated data (n = 3). Participants who ingested at least 210 g/week of alcohol (males) (n = 1) and 140 g/week (females) (n = 2) were also excluded. Subjects with viral hepatitis and cryptogenic fatty liver were also excluded (n = 2). Ultimately, 288 participants were included in this investigation (Figure 1).

Measurement of Hepatic Steatosis and Hepatic Fibrosis

MAFLD is defined as one of the following conditions when there is fatty deformation in the liver: (1) BMI ≥23 kg/m^2 (Asian population), or WC >94 cm (males) and >80 cm (females); (2) Fasting blood glucose level ≥5.6 mmol/L (100 mg/dL), or blood glucose level ≥7.8 mmol/L (≥140 mg/dL) after 2 h of sugar loading, or glycated hemoglobin A1c (HbA1c) ≥5.7% (39 mmol/L), or T2DM or treatment of T2DM; (3) Blood pressure ≥130/85 mmHg or specific antihypertensive drug treatment. (4) Plasma triglyceride (TG) ≥1.70 mmol/L (150 mg/dL) or lipid-lowering treatment. (5) Plasma high-

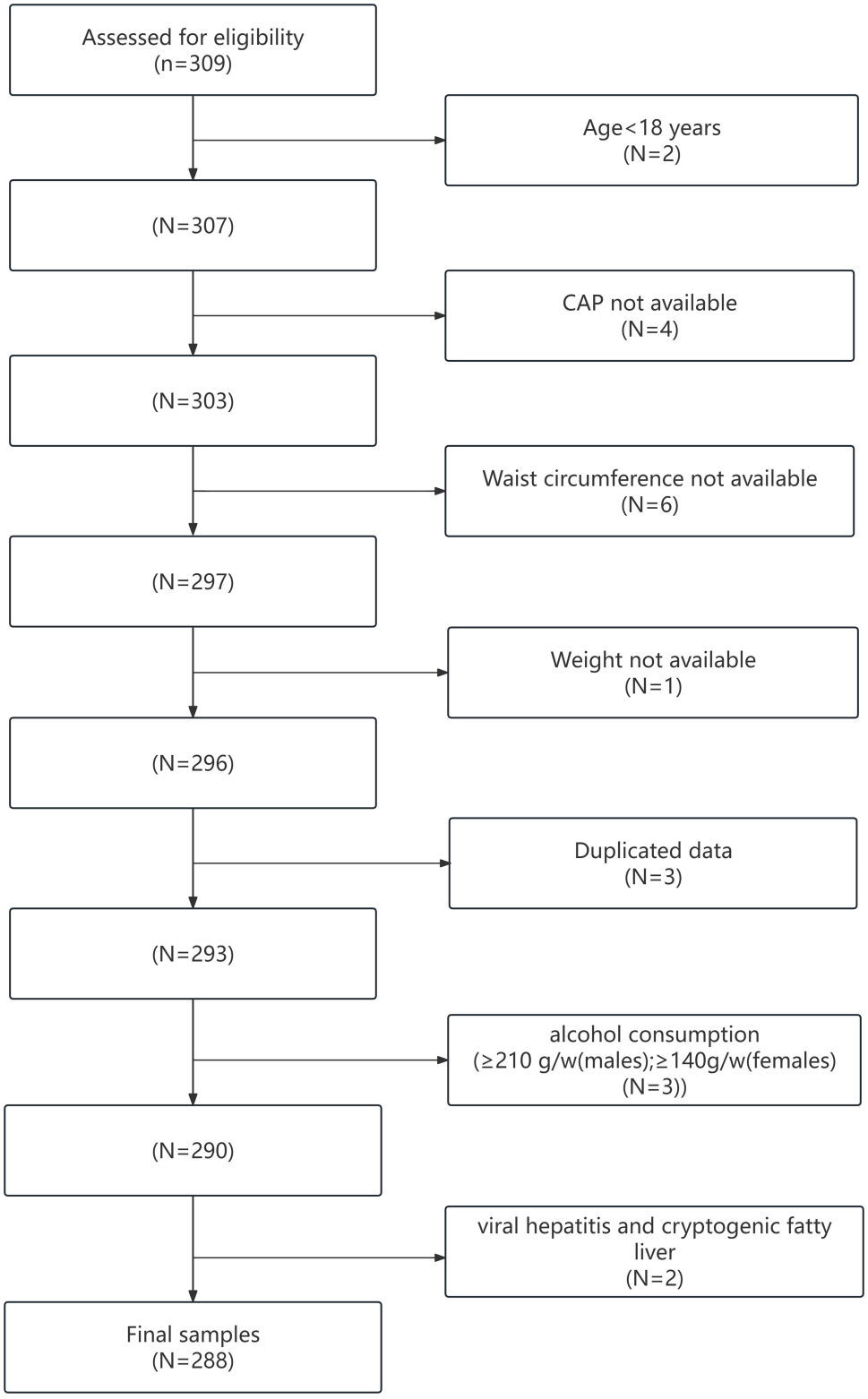

Figure 1 Flowchart of the sample selection from outpatient patients at the Affiliated Hospital of Hangzhou Normal University.

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density lipoprotein cholesterol (HDL-C) ≤1.0 mmol/L (40 mg/dL) in males, ≤1.3 mmol/L (50 mg/dL) in females, or lipid-lowering treatment.[22] In addition, hepatic steatosis and hepatic fibrosis were assessed among participants using the FibroScan® machine, measuring the CAP and liver stiffness measurements (E values). A median CAP value ≥274 dB/m was considered indicative of hepatic steatosis,[23] while an E value ≥8.0 kPa signaled hepatic fibrosis.[24] These thresholds were utilized to identify crucial parameters associated with MAFLD within the study cohort.

Definition of WWI

WWI is a measure of abdominal obesity and is calculated as the WC (cm) divided by the square root of body weight (kg), expressed as cm/kg2.[17,19] WC and weight measurements were obtained by trained health technicians. Body weight was measured by a digital weight scale with a precision of 0.1 kg, while WC was measured by a retractable tape at the end of exhalation, with a precision of 0.1 cm.

WWI scores were divided into quartiles (quartile 1–quartile 4), with respective ranges of 7.14–10.16, 10.16–10.57, 10.57–11.10, and 11.10–12.58, respectively, and each quartile comprising 72 participants.

Variables

Based on previous studies, the following variables were screened to investigate the effect of multiple variables on the relationship between WWI and hepatic steatosis and hepatic fibrosis.[15,23,25–30] Table 1 presents the variables along with their respective details.

Covariates for demographic and past medical history, such as age, sex, height, WC, weight, hypertension, T2DM, cardiovascular disease (CVD), alcohol consumption, and smoking, were collected from the Affiliated Hospital of

Table 1 Characteristics of the Participants

Characteristics	Q1 (N=72) 7.14~10.16	Q2 (N=72) 10.16~10.57	Q3 (N=72) 10.57~11.10	Q4 (N=72) 11.10~12.58	P value
Age(years),(%)					0.000
18~39 40~59 ≥60	45 (62.5%) 21 (29.2%) 6 (8.3%)	30 (41.7%) 32 (44.4%) 10 (13.9%)	15 (22.7%) 50 (69.4%) 7 (9.7%)	16 (22.2%) 41 (56.9%) 15 (20.8%)	
Sex (male/female),(%)					0.110
Male Female	44 (61.1%) 28 (38.9%)	51 (70.8%) 21 (29.2%)	49 (68.1%) 23 (31.9%)	38 (52.8%) 34 (47.2%)	
BMI (kg/m^2),(%)					0.000
≤18.5 1.8~24 24~28 >28	6 (8.3%) 33 (45.8%) 24 (33.3%) 9 (12.5%)	1 (1.4%) 32 (44.4%) 32 (44.4%) 7 (9.7%)	0 (0%) 12 (16.7%) 39 (54.2%) 21 (29.2%)	2 (2.8%) 22 (30.6%) 31 (43.1%) 17 (23.6%)	
Smoking,(%)					0.246
Never Presence Quitting	56 (77.8%) 16 (22.2) 0 (0%)	54 (75.0%) 14 (19.4) 4 (5.6%)	46 (63.9%) 20 (27.8) 6 (8.3%)	52 (72.2%) 15 (20.8%) 5 (6.9%)	
Alcohol consumption,(%)					0.845
Never or small (<40 g/w) Light or above (≥40 g/w)	23 (31.9%) 49 (68.1%)	25 (34.7%) 47 (65.3%)	23 (31.9%) 49 (68.1%)	20 (27.8%) 52 (72.2%)	

(*Continued*)

Table 1 (Continued).

Characteristics	Q1 (N=72) 7.14~10.16	Q2 (N=72) 10.16~10.57	Q3 (N=72) 10.57~11.10	Q4 (N=72) 11.10~12.58	P value
Hypertension,(%)					0.077
Yes No	31 (43.1%) 41 (56.9%)	34 (47.2%) 38 (52.8%)	45 (62.5%) 27 (43.1%)	41 (56.9%) 31 (43.1%)	
T2DM,(%)					0.876
Yes No	10 (13.9%) 62 (86.1%)	11 (15.3%) 61 (84.7%)	13 (18.1%) 59 (81.9%)	13 (18.1%) 59 (81.9%)	
CVD,(%)					0.080
Yes No	7 (9.7%) 65 (90.3%)	14 (19.4%) 58 (79.7%)	10 (13.9%) 62 (86.1%)	18 (25.0%) 54 (75.0%)	
MAFLD,(%)					<0.001
Yes No	31 (43.1%) 41 (56.9%)	47 (65.3%) 25 (34.7%)	52 (72.2%) 20 (27.8%)	55 (76.4%) 17 (23.6%)	
WC (cm)	79.42±9.33	85.56±7.31	92.90±7.02	95.18±8.94	<0.001
Height (cm)	168.91±8.13	168.11±8.50	166.27±6.62	162.89±7.76	<0.001
Weight (kg)	67.66±13.07	68.82±11.40	73.84±10.72	68.49±13.16	0.010
Laboratory feature					
TC (mmol/L)	4.95±1.15	4.58±1.03	4.92±1.09	4.92±1.25	0.157
TG (mmol/L)	1.48±1.12	1.55±0.88	2.09±1.84	2.04±2.51	0.059
ALT (IU/L)	21.79±10.67	31.51±19.19	33.15±24.18	30.51±19.29	0.002
AST (IU/L)	20.92±5.89	24.73±15.91	23.33±10.07	28.38±33.77	0.141
SUA (μmol/L)	353.99±90.31	364.82±80.46	365.72±100.22	357.23±104.37	0.849
E value (kPa)	5.11±2.93	5.09±1.56	5.47±1.75	5.75±2.98	0.293
HDL-C (mmol/L)	1.29±0.30	1.16±0.24	1.15±0.26	1.22±0.35	0.018
LDL-C (mmol/L)	2.90±0.84	2.77±0.72	2.89±0.74	2.98±0.87	0.470
CAP (dB/m)	248.43±47.85	267.94±47.22	281.56±50.81	282.05±47.07	0.000

Note: Values were expressed as mean±SD or n (%).

Abbreviations: BMI, body mass index; CVD, cardiovascular disease; MAFLD, metabolic disease-associated fatty liver disease; WC, waist circumference; TC, total cholesterol; TG, Triglyceride; ALT, alanine aminotransferase; AST, aspartate aminotransferase; SUA, serum uric acid; E value, liver hardness measurement; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; CAP, controlled attenuation parameter; WWI, weight-adjusted waist index.

Hangzhou Normal University. Alcohol consumption status was categorized as never or small drinking (<40 g/w), and light or above drinking (≥40 g/w). Additionally, BMI was calculated as body weight divided by the square of height, expressed in kg/m2.[31] Furthermore, laboratory analyses included fasting elbow venous blood (3 mL) measurements of total cholesterol (TC, mmol/L), triglyceride (TG, mmol/L), aspartate aminotransferase (AST, IU/L), alanine aminotransferase (ALT, IU/L), serum uric acid (SUA, μmol/L), high-density lipoprotein cholesterol (HDL-C, mmol/L), and low-density lipoprotein cholesterol (LDL-C, mmol/L).

Statistical Analysis

All variables were summarized in union descriptive statistics, expressing continuous variables as mean ±standard deviation and dichotomized variables as values and percentages. Differences between WWI quartiles were assessed using chi-square tests, and single-factor ANOVA tests, while the Kruskal Wallis test was performed for non-normally distributed data. Sensitivity analysis was used to evaluate the robustness of WWI quartiles. Multiple linear regression analysis encompassing three models was employed to evaluate the relationship between WWI and hepatic steatosis and hepatic fibrosis. Notably, Model 1: unadjusted; Model 2: adjusted for age and gender; Model 3: adjusted for age, sex, BMI, hypertension, diabetes, CVD, alcohol consumption, smoking, TC, TG, ALT, AST, SUA, HDL-C, and LDL-C. We used a stratified multivariate logistic regression model to analyze the relationship between WWI and hepatic steatosis and hepatic fibrosis in subgroups. The covariates covered in the subgroup analysis included age, alcohol consumption, BMI, CVD, T2DM, sex, hypertension, and smoking. Statistical analyses were conducted using the SPSS software, with $P \leq 0.05$ reflecting a statistically significant difference. This comprehensive approach ensures a rigorous exploration of the relationships under consideration.

Result

Participant Characteristics

Following rigorous screening, a total of 288 participants were included in this study. Table 1 illustrates the relationship between baseline data and WWI quartiles. All participants (182 males and 106 females) were of age ≥18 years. As WWI increased, a gradual rise in male participants and BMI and WC levels was observed ($P < 0.05$). Notably, a decrease in HDL-C levels was observed with increasing WWI ($P < 0.05$) (Table 1).

Association Between the WWI and CAP

Across all three models, a positive linear correlation was observed between WWI and hepatic steatosis ($P < 0.05$). Upon adjusting for all covariates in Model 3, each one-unit increase in WWI score corresponded to a 73.64 dB/m increase in CAP ($\beta = 73.64$; 95% CI 4.42; 10.78; $P < 0.001$). Furthermore, quartile analysis of WWI revealed a significant relationship between WWI and hepatic steatosis. In Model 1, the β values of CAP in Q2, Q3, and Q4 were 19.51, 32.61, and 33.62, respectively ($P < 0.05$). Similarly, in Model 2, the β-values of CAP in Q2, Q3, and Q4 were 19.59, 33.78, and 38.8, respectively, when compared to the lower quartile (Q1) ($P < 0.05$) (Table 2).

Table 2 The Association Between WWI with CAP and E-Value

WWI	Crude Model (Model 1)[a]	Minimally Adjusted Model (Model 2)[b]	Fully Adjusted Model (Model 3)[c]
CAP β (95% CI) [d]	248.43 (237.22,259.63) p<0.000	287.69 (263.03,312.36) p<0.000	73.64 (15.97,131.31) p<0.013
WWI group			
Quartile 1	0	0	0
Quartile 2	19.51 (3.66,35.36) p=0.016	19.59 (3.65,35.52) p=0.016	9.68 (−4.17,23.54) p=0.170
Quartile 3	32.61 (16.71,48.51) p<0.000	33.78 (17.51,50.06) p<0.000	7.24 (−7.70,22.19) p=0.341
Quartile 4	33.62 (17.77,49.47) p<0.000	38.85 (22.34,55.36) p<0.000	14.67 (−0.24,29.59) p=0.054
P for trend			

(*Continued*)

Table 2 (Continued).

WWI	Crude Model (Model 1)[a]	Minimally Adjusted Model (Model 2)[b]	Fully Adjusted Model (Model 3)[c]
E value β (95% CI)	5.11 (4.56,5.67) p<0.000	5.07 (3.82,6.32) p<0.000	−0.15 (−3.38,3.08) p=0.928
WWI group			
Quartile 1	0	0	0
Quartile 2	−0.02 (−0.81,0.76) p=0.959	−0.22 (−1.03,0.58) p=0.581	−0.61 (−1.39,0.16) p=0.119
Quartile 3	0.33 (−0.45,1.12) p=0.403	0.08 (−0.73,0.91) p=0.835	−0.51 (−1.35,0.32) p=0.226
Quartile 4	0.63 (−0.15,1.42) p=0.113	0.42 (−0.413,1.25) p=0.320	−0.14 (−0.97,0.69) p=0.741
P for trend			

Notes: In the sensitivity analysis, the weight-adjusted waist circumference index was converted from a continuous variable to a categorical variable (quartiles).[a]Model 1: Unadjusted for covariates [b]Model 2: Adjusted for sex and age [c]Model 3: Adjusted for sex, age, height, WC, weight, hypertension, T2DM, CVD, alcohol consumption, smoking, TC, TG, ALT, AST, SUA, HDL-C, and LDL-C [d]95% CI: 95% confidence interval.

Association Between the WWI and E Value

Model 1 demonstrated that the E value increases by 5.11 kPa for each unit of WWI score (β = 5.11; 95% CI: 4.56, 5.67; P < 0.000). Similarly, in Model 2, For every unit increase in WWI score the E value increases by 5.07 kPa (β = 5.07; 95% CI: 3.82, 6.32; P < 0.000). However, when considering the WWI quartile, no statistically significant positive association between WWI and hepatic fibrosis was observed across all models (P > 0.05) (Table 2).

Subgroup Analysis

Subgroup analyses were conducted to explore potential variations in the correlation between WWI with CAP/E values across different population characteristics. Stratified multivariate regression analyses were performed for various demographic factors including age groups, alcohol consumption, BMI, CVD, T2DM, sex, hypertension, and smoking (Figures 2 and 3).

Notably, a stronger positive correlation between WWI and CAP was observed in smokers and individuals aged 18–59 years (P < 0.05) (Figure 2). Similarly, drinking, male, with BMI >28, and participants with T2DM exhibited a positive correlation between WWI and E values (P < 0.05) (Figure 3).

Discussion

Given the global impact and escalating risks associated with MAFLD, there is an urgent need for early screening and vigilant monitoring of this condition. Previous studies underscore the significance of BMI, WC, waist-to-height ratio, and triglyceride-glucose index in identifying MAFLD risk.[32–34] Building upon this foundation, we investigated the connection between WWI and MAFLD. This study demonstrated a significant linear correlation between WWI and hepatic steatosis and fibrosis. In models 1, 2 and 3, for every unit of WWI score increase, the CAP value increases by 248.43 dB/m, 287.69 dB/m and 73.64 dB/m respectively. When considering the WWI quartile, there is a significant relationship between WWI and hepatic steatosis. In models 1, 2, for every unit of WWI score increase, the E value increases by 5.11 kPa and 5.07kPa respectively. Subgroup analysis further showed that WWI was significantly and positively associated with hepatic steatosis in smokers and individuals aged 18–59 years, whereas WWI was positively associated with hepatic fibrosis in participants who consumed alcohol, were male, had a BMI >28, and had T2DM.

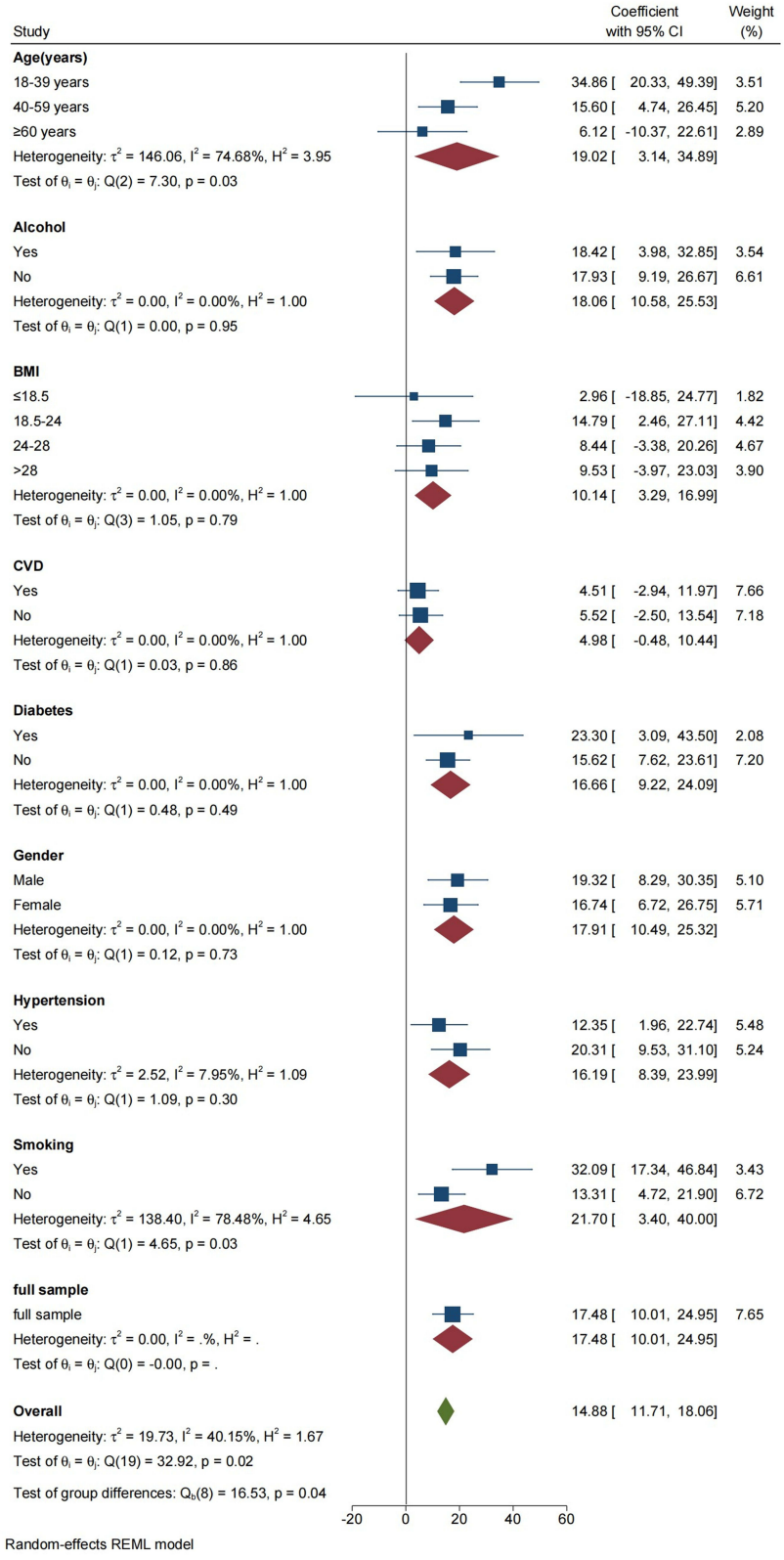

Figure 2 Subgroup analysis for the association between WWI and hepatic steatosis.

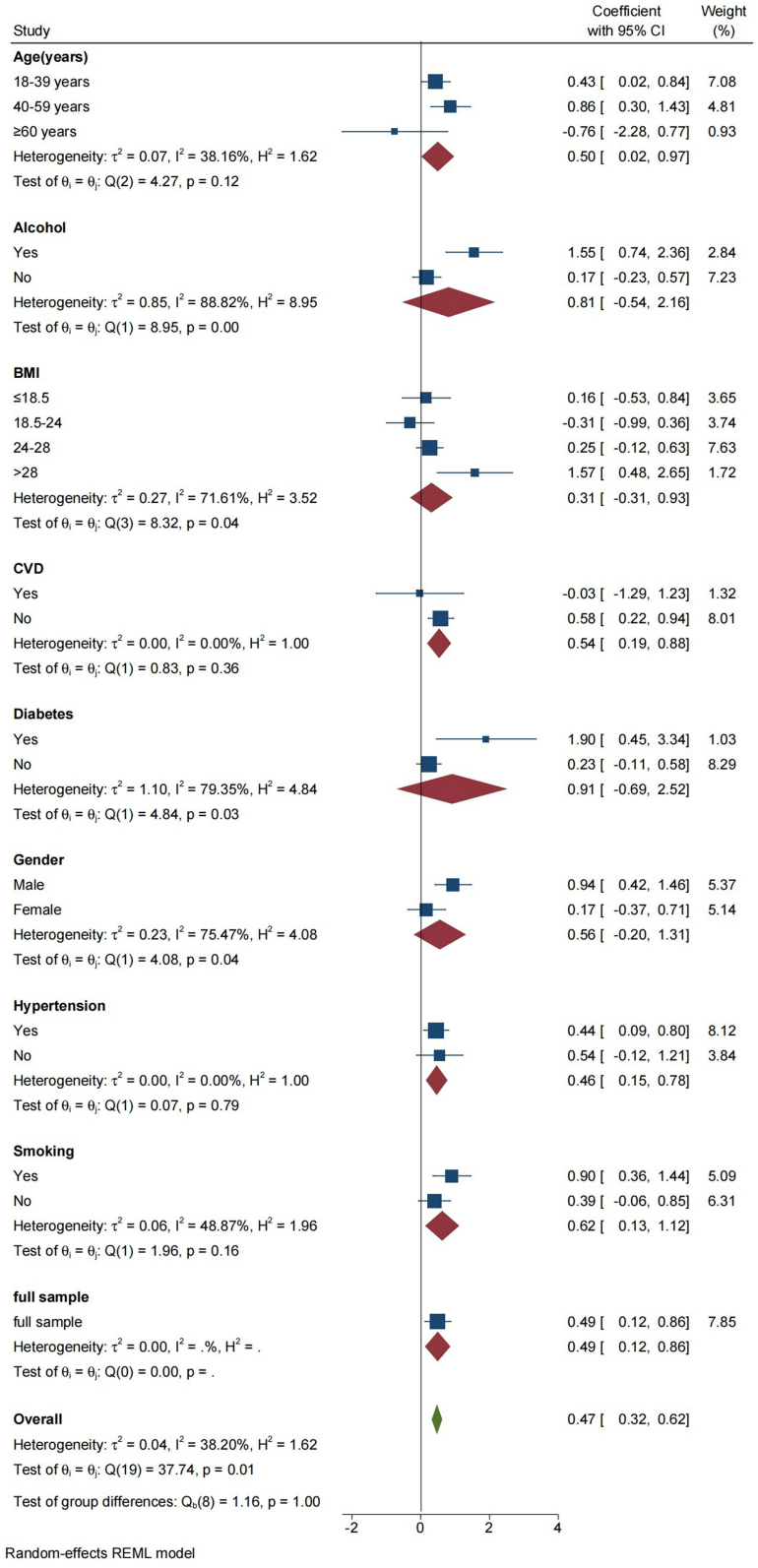

Figure 3 Subgroup analysis for the association between WWI and hepatic fibrosis.

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Characterized by hepatic lipid accumulation and metabolic disturbances.[22] The incidence of MAFLD has risen steadily worldwide in recent years.[35] Lipid metabolism disorders are in turn closely related to obesity.[4] Previous studies have established the contribution of increased BMI and WC to the degree of fibrosis in MAFLD.[15] While BMI and WC are conventional metrics for obesity evaluation.[31] It is crucial to recognize that obesity encompasses excess adipose tissues and not just weight gain.[36] BMI cannot distinguish between muscle tissues and adipose tissues.[37–39] WC is also an index that depends on the overall body weight.[17] Conversely, WWI, derived from standardized body weight, offers a comprehensive assessment inclusive of both muscle and fat components. This measure accurately reflects high-fat mass and low muscle mass, while remaining insensitive to overall body weight fluctuations.[19] A large number of studies have shown a positive correlation among WWI and T2DM, hyperuricemia, CVD (arteriosclerosis), and high risk of all-cause death, and WWI is positively correlated with hyperuricemia in patients with hypertension, negative correlation between SUA levels and coronary artery collateral opening. However, research on the relationship between WWI and MAFLD remains limited.[17,40–43] Given that MAFLD is mainly characterized by abdominal obesity and visceral fat accumulation, and considering WWI's positive correlation with abdominal fat and visceral fat.[18,44,45] This study delved into the correlation between WWI and MAFLD to provide additional evidence for the prevention and screening of MAFLD. This analysis showed that WWI had a significant linear positive correlation with hepatic steatosis and fibrosis, underscoring the key role of abdominal obesity in the development of hepatic steatosis. Furthermore, WWI holds promise as a simple and effective indicator for early MAFLD screening.

Analyzing MAFLD characteristics and its relationship with metabolic disorders underscores the close relationship between MAFLD development and abdominal obesity, skeletal muscle loss, and significant visceral fat accumulation.[44–46] This association is attributed to the anatomical proximity of visceral adipose tissue to the liver, facilitating the breakdown of visceral fat into non-esterified fatty acids (NEFA). NEFA can induce liver metabolic disorders, including glucose intolerance, dyslipidemia, hyperinsulinemia, and insulin resistance. Despite the potential of insulin to enhance NEFA re-lipidation, visceral fat resistance toward this process.[47] In addition, liver disease can promote an inflammatory response.[48] This conclusion not only emphasizes the correlation between visceral adiposity, insulin resistance, and hepatic inflammation but also supports our finding of a positive correlation between WWI and MAFLD.

This study possesses several advantages compared to previous studies on the relationship between WWI and MAFLD. This is the first study to use the new definition of MAFLD to analyzed patient data from the Affiliated Hospital of Hangzhou Normal University in China. Through rigorous adjustment for various potential covariates, a positive correlation between WWI and MAFLD was revealed, which is consistent with previous research results. In addition, through subgroup analysis, a stronger positive association between WWI and hepatic steatosis was found for the first time among smokers. Importantly, while previous studies by Shen et al found WWI's relationship with hepatic fibrosis to be non-linear, and with hepatic steatosis to be linear, Hu et al found the opposite.[20,21] This study demonstrates a linear positive correlation between WWI and MAFLD. This discrepancy could be attributed to population-specific characteristics captured in the study, bringing this findings closer to the characteristics of the local population.

Furthermore, subgroup analysis identified specific demographics with significant correlations between WWI and MAFLD, providing tailored clinical recommendations. Notably, there was a significant correlation between WWI and CAP for participants aged 18–59 years, compared with those aged >60 years. A study based on the Chinese population reported that the fat mass and visceral fat level increased in the 55–65-age group, but decreased in the 70–85-age group, which indicates that the younger population has higher fat mass and visceral fat levels.[49] This may be due to the differences in fat mass and visceral fat among different races and cultures. Moore et al observed that people in China aged >65 years tend to have a healthier lifestyle.[50] Notably, among smokers, a stronger positive correlation was observed between WWI and CAP. In addition, drinking, male gender, BMI >28, and participants with T2DM exhibited a positive relationship between WWI and hepatic fibrosis, which is consistent with established influences on MAFLD.[4,51–54]

However, this study has limitations. First, MAFLD diagnosis relied solely on CAP and E values, without liver biopsy confirmation. Second, the cross-sectional study design itself cannot determine the causal relationship between WWI and MAFLD. Third, Although we include covariates as much as possible, the development of hepatic steatosis and hepatic fibrosis involves many potential factors, so there may still be unmeasured confounding factors, then, only individuals aged ≥18 years were included, which limits generalizability to those aged <18 years. Finally, the study focused on

patients from the affiliated hospital of hangzhou normal university, potentially limiting the applicability of our findings to other geographic regions or ethnic populations.

Conclusion

In this study, a significant positive correlation was found between WWI and MAFLD. Specifically, individuals aged 18–59 years and smokers should be particularly vigilant regarding hepatic steatosis development. Moreover, individuals who drink, males, those with a BMI >28, and participants with diabetes should closely monitor the progression to hepatic fibrosis. These findings are of great significance to the prevention and intervention of MAFLD, and the follow-up study can further explore the relationship between WWI and MAFLD in populations with high-risk characteristics. Moving forward, given that WWI is an easily measurable indicator, we propose that WWI could serve as a simple and effective potential indicator for the early screening of MAFLD.

Data Sharing Statement

All study data can be requested from the corresponding author.

Consent for Publication

All authors agree to publication.

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Disclosure

The authors declare that they have no competing interests in this work.

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