Ren et al. Journal of Translational Medicine (2025) 23:859
https://doi.org/10.1186/s12967-025-06870-z

Journal of
Translational Medicine

RESEARCH

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The role of irregular eating behaviors in metabolic dysfunction-associated steatotic liver disease: evidence from a multicenter cross-sectional study in China

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Abstract

Background Irregular eating patterns, such as skipping breakfast and late dinner consumption, have been linked to adverse metabolic profiles. However, the impact of these behaviors or their combined effect on metabolic dysfunction-associated steatotic liver disease (MASLD) remains unclear.

Methods In this multicenter cross-sectional study, we analyzed data from 1965 adults recruited from diabetes, obesity, and metabolic disease clinics in six municipalities/provinces in China. Participants were categorized for analysis based on two irregular eating habits assessed by monthly frequency: skipping breakfast and late dinner consumption. For individual analyses, each habit was treated as a binary variable, dividing participants into two groups (e.g., skippers vs. non-skippers). For the combined analysis, these habits were aggregated to form a three-level ordinal variable, which classified participants based on the number of habits they reported: "No" (zero), "Medium" (one), or "Severe" (both). We employed logistic regression models to calculate the odds ratios (ORs) for MASLD associated with skipping breakfast, late dinner consumption, and their combination, after adjusting for key demographic, lifestyle, and metabolic factors.

Results The prevalence of MASLD was higher among breakfast skippers compared to those who ate breakfast regularly (52.9% vs. 47.2%, $p=0.023$), and similarly higher among late dinner eaters compared to those with regular dinner times (54.4% vs. 46.8%, $p=0.003$). In the combined analysis, a graded relationship was observed across the three-level ordinal variable, with MASLD prevalence increasing from 46.5% in the "No" irregular eating group, to 50.3% in the "Medium" irregular eating group, and 55.5% in the "Severe" irregular eating group ($p=0.01$). After multivariable adjustment, skipping breakfast, late dinner consumption, and the combination of these behaviors were each

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independently associated with higher odds of MASLD. This was highlighted by a clear dose–response relationship for the combined behaviors, where the odds of MASLD systematically increased with the severity of irregular eating (from "No" to "Medium" to "Severe"). Notably, the "Severe" irregular eating group, representing those with both habits, had a 65% higher odds of MASLD compared to those with none (OR = 1.65, 95% CI 1.17–2.34, $p = 0.004$).

Conclusions Our study indicates that irregular eating behaviors, such as skipping breakfast and late dinner consumption, as well as their combination, are significantly associated with a higher prevalence of MASLD.

Keywords Irregular eating behaviors, Skipping breakfast, Late dinner consumption, Metabolic dysfunction-associated steatotic liver disease

Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) has emerged as a widespread global health concern, closely interrelated with obesity, type 2 diabetes, and metabolic syndrome [1]. As a major cause of chronic liver conditions, MASLD significantly increases the risk of cardiovascular events, cirrhosis, and hepatocellular carcinoma [2–4]. Lifestyle interventions and bariatric surgery, which aim to achieve significant weight loss, are key components of MASLD treatment, as they improve histological outcomes and reduce the risk of comorbidities [5]. Identifying modifiable lifestyle factors is essential for addressing the growing burden of MASLD.

Emerging evidence suggests that irregular meal patterns may contribute to metabolic dysfunction [6, 7]. Our previous research discovered that frequent rapid eating was associated with a higher risk of MASLD [8]. The Chinese Nutrition Society reveals that 35% of respondents fail to eat breakfast daily, while 11% skip breakfast regularly. Skipping breakfast, for example, has been associated with impaired glucose tolerance, increased body weight, and unfavorable lipid profiles [9, 10]. Late dinner consumption, frequently accompanied by reduced overnight fasting intervals, may disrupt normal metabolic cycles, leading to weight gain and insulin resistance [11]. Moreover, animal studies were also carried out to explore the effects of meal time and unhealthy dietary habits, such as skipping breakfast and eating late at night, on metabolic health [12, 13]. Although each of these behaviors has been individually associated with metabolic disturbances, research exploring their individual or combined effect on MASLD remains limited. This gap highlights the need for comprehensive studies to understand how the combination of irregular meal patterns, such as skipping breakfast and consuming late dinners, may collectively influence the development of MASLD.

In this multicenter cross-sectional study, we investigated the association of skipping breakfast, late dinner consumption, and their combination with the prevalence of MASLD in a cohort of Chinese adults attending diabetes, obesity, and metabolic disease clinics located in six municipalities/provinces in China. We hypothesized that participants who engaged in both behaviors frequently would have a higher probability of MASLD compared to individuals with less frequent or mixed patterns. Understanding the cumulative impact of these irregular eating patterns can guide recommendations to improve meal timing and potentially reduce MASLD risk.

Methods

Study design and population

A total of 2704 participants were recruited for this study from 10 clinics specializing in diabetes, obesity, and metabolic diseases across six municipalities/provinces in China between January and December 2011, as described in our previous research [14]. In order to minimize selection bias, an online patient registration system was employed and a distinctive clinic ID for each participant was documented to guarantee continuous enrolment and prevent the repeated input of individual records. Participants were excluded if they lacked key information on dietary habits or clinical assessments, consumed alcohol above the predefined thresholds (> 10 g per day for women and > 20 g per day for men), or had viral hepatitis, other known liver diseases. After the application of these criteria, a total of 1965 eligible individuals were encompassed in the final analysis.

Clinical and laboratory measurements

Based on the Qinling-Huaihe Line, which is the primary geographical divide between North and South China, participants were grouped into Northern (Tianjin, Shandong, and Heilongjiang) and Southern (Henan, Shanghai, and Jiangsu) regions. While Henan province is geographically positioned at the boundary between North and South China, with most of its territory located north of the Qinling-Huaihe Line, its classification into the Southern group was based on prior evidence demonstrating closer genetic and metabolic similarities of its population to Southern Han Chinese cohorts [14, 15]. Information regarding age, sex, region, alcohol-drinking status, and smoking status was gathered through standardized questionnaires. Standardized protocols were employed to measure anthropometric parameters including weight, height, and waist circumference, as well as blood pressure. Body mass index (BMI) was calculated as weight (kg) divided by squared height (m^2). Fasting blood samples were obtained to determine glucose, lipid profiles, and liver enzymes. Following the results of a 75 g oral glucose tolerance test, the participants were classified into three groups according to their glucose metabolism status: normal glucose tolerance (NGT), impaired glucose regulation (IGR), and type 2 diabetes mellitus (T2DM). Fatty liver was diagnosed based on the same criteria used for hepatic steatosis as determined by ultrasonography [14, 16].

Assessment of irregular eating behaviors

Standardized questionnaires administered by trained interviewers were used to evaluate two dietary patterns: breakfast skipping and late dinner consumption. Participants who reported engaging in either behavior one time per month or less were classified as having no habitual irregularity for that specific behavior. These dichotomous classifications were then combined to create a comprehensive three-level composite measure of irregular eating patterns. The "no irregular eating behaviors" category included participants with neither skipping breakfast, nor late dinner consumption, while the "medium irregular eating behaviors" category encompassed those with either skipping breakfast or late dinner consumption. The "severe irregular eating behaviors" category comprised participants who exhibited both skipping breakfast and late dinner consumption. This ordinal classification system provides a structured approach to operationalizing graded exposure to meal timing irregularities, where each successive level represents increasing cumulative behavioral intensity and greater departure from regular eating patterns.

Statistical analysis

Continuous variables were presented as means ± standard deviations or medians along with the interquartile range (25%–75%), while categorical variables were shown as percentages. The independent-samples t-test or Mann–Whitney U test was employed to compare the differences between groups of data. Differences between groups were assessed by using one-way ANOVA or Kruskal–Wallis tests for continuous variables and chi-square tests for categorical variables. Logistic regression models were subsequently constructed to examine the association between the irregular eating behavior variable and MASLD. All statistical analyses were conducted using standard statistical software (R version 4.2.3), with statistical significance defined as a two-sided *p*-value of less than 0.05.

Results

General characteristics of participants in different groups

Among the 1965 participants in this analysis, the average age was in the mid-fifties, and the gender distribution was almost balanced between males and females (Table 1). In contrast, participants in the "Severe" irregular eating group were significantly younger, with a median age of 50 years. Despite being younger, this group also had the highest liver fat content. We found a significant dose–response relationship between the cumulative number of irregular eating behaviors and MASLD prevalence ($p = 0.01$). The proportion of individuals with MASLD rose from 46.5% in the group with no habits ("No" irregular eating group) to 50.3% for one habit ("Medium" irregular eating group) and 55.5% for both ("Severe" irregular eating group). As shown in Table 1, participants who reported skipping breakfast and having late dinners were found to have certain lifestyle characteristics. Specifically, they were more likely to be alcohol drinkers ($p = 0.004$) and smokers ($p = 0.001$), suggesting a potential association between irregular eating behaviors and these habits. Additionally, these individuals were more likely to come from the Northern region of China ($p < 0.001$), indicating a regional pattern in the prevalence of these behaviors. This highlights the complex interplay between dietary habits, lifestyle choices, and geographic factors. Individuals with more severe irregular eating behaviors exhibited less favorable metabolic profiles, including higher glycated hemoglobin A1c (HbA1c) levels and lower high-density lipoprotein cholesterol (HDL-C), compared to those with milder irregular eating behaviors.

As illustrated in Fig. 1A, participants with breakfast-skipping habits demonstrated a higher MASLD prevalence (52.9%) compared to those without this habit (47.2%). Correspondingly, Fig. 1B reveals that individuals habitually consuming late dinners exhibited elevated MASLD rates (54.4%) versus non-habitual consumers (46.8%). These consistent patterns in both dietary behaviors indicate that habitual irregularities in meal timing, whether it is skipping breakfast or having a late dinner, are associated with a systematically higher prevalence of MASLD.

Stepwise logistic regression analysis of associations with MASLD

Table 2 presents a stepwise logistic regression analysis of the association between breakfast skipping and MASLD. After adjusting for age, sex, smoking, alcohol consumption, BMI, and region, the odds ratio (OR) for breakfast skippers increased to 1.37 (95% CI 1.09–1.72; $p = 0.007$). Further adjustment for additional confounders strengthened this association (OR = 1.58, 95% CI 1.20–2.11,

Table 1 Baseline characteristics of study participants stratified by irregular eating behaviors

Characteristics	Categories of irregular eating behaviors			*p*
	No (n = 1241)	Medium (n = 380)	Severe (n = 344)	
MASLD, n (%)	577 (46.5)	191 (50.3)	191 (55.5)	0.01
North China, n (%)	491 (39.6)	211 (55.5)	194 (56.4)	< 0.001
Women, n (%)	644 (51.9)	177 (46.6)	167 (48.5)	0.15
Age (year)	54.00 (45.00, 62.00)	54.00 (43.00, 62.00)	50.00 (39.00, 60.00)*	< 0.001
Smoker, n (%)	307 (24.7)	122 (32.1)	113 (32.8)	0.001
Alcohol-drinker, n (%)	252 (20.3)	99 (26.1)	95 (27.6)	0.004
BMI (kg/m^2)	24.41 (22.27, 26.78)	24.69 (22.75, 26.81)	24.61 (22.72, 26.98)	0.678
SBP (mmHg)	130 (120, 140)	128 (119, 140)*	126 (120, 140)*	0.007
DBP (mmHg)	80.00 (73.00, 88.00)	80.00 (70.00, 86.00)	80.00 (72.00, 87.25)	0.282
Glucose metabolism, n (%)				0.727
NGT	314 (25.3)	89 (23.4)	91 (26.5)	
IGR	178 (14.3)	61 (16.1)	44 (12.8)	
T2DM	749 (60.4)	230 (60.5)	209 (60.8)	
FBG (mmol/L)	6.11 (5.27, 7.80)	6.10 (5.25, 7.70)	6.30 (5.24, 8.59)	0.305
2hPBG (mmol/L)	11.40 (6.90, 16.50)	11.35 (7.23, 16.17)	11.82 (7.00, 17.67)	0.47
HbA1c (%)	6.80 (5.80, 8.70)	7.00 (5.90, 8.85)	7.55 (5.90, 9.67)*	0.004
TC (mmol/L)	4.93 (4.27, 5.73)	4.71 (3.97, 5.45)*	4.63 (3.84, 5.47)	< 0.001
TG (mmol/L)	1.50 (1.03, 2.27)	1.44 (0.96, 2.18)	1.47 (0.99, 2.21)	0.764
HDL-C (mmol/L)	1.20 (1.00, 1.50)	1.16 (0.95, 1.40)	1.11 (0.93, 1.34)	< 0.001
LDL-C (mmol/L)	2.82 (2.30, 3.46)	2.74 (2.21, 3.30)*	2.65 (2.11, 3.22)*	0.002
ALT (U/L)	17.85 (12.00, 26.00)	20.00 (13.00, 28.00)*	20.00 (14.00, 30.00)	< 0.001
AST (U/L)	20.54 (16.00, 26.00)	19.00 (15.00, 25.00)	21.00 (16.00, 29.00)#	0.06
GGT (U/L)	24.00 (15.00, 43.25)	29.00 (18.00, 49.00)*	38.00 (19.44, 59.00)#	< 0.001
UA (μmol/L)	288.00 (227.00, 355.00)	293.50 (228.25, 363.00)	283.48 (219.00, 345.00)	0.182
LFC, %	13.53 (7.85, 22.54)	14.43 (8.44, 23.13)	15.93 (8.74, 24.79)*	0.042

MASLD metabolic dysfunction-associated steatotic liver disease, *BMI* body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *NGT* normal glucose tolerance, *IGR* impaired glucose regulation, *T2DM* type 2 diabetes mellitus, *FBG* fasting blood glucose, *2hBG* 2-h postprandial blood glucose, *HbA1c* glycated hemoglobin A1c, *TC* total cholesterol, *TG* triglyceride, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol, *ALT* alanine aminotransaminase, *AST* aspartate aminotransferase, *GGT* gamma-glutamyl transferase, *UA* uric acid, *LFC* liver fat content

* Indicates a statistically significant difference compared to the No irregular eating group ($p < 0.05$)

# Indicates a statistically significant difference compared to the Medium irregular eating group ($p < 0.05$)

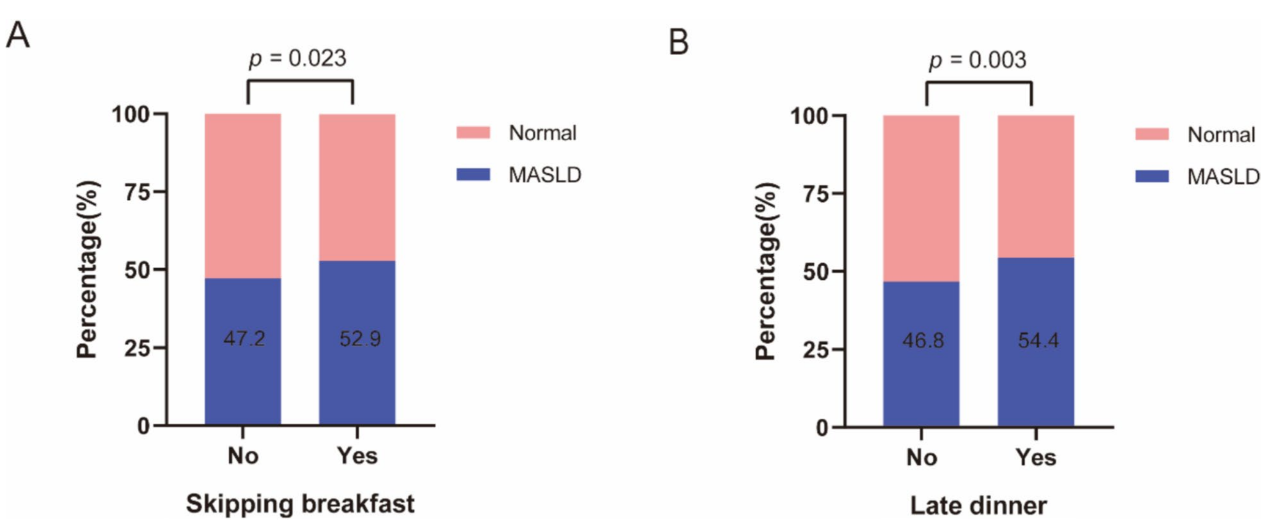

Fig. 1 MASLD prevalence based on skipping breakfast and late dinner behaviors

Table 2 Logistic regression analysis of the association between skipping breakfast and MASLD

Logistic regression models	Categories of skipping breakfast	
	No	Yes
Unadjusted ORs (95%CIs)	Reference	1.26 (1.03–1.53)
p value		0.02
Model 1 ORs (95%CIs)	Reference	1.37 (1.09–1.72)
p value		0.007
Model 2 ORs (95%CIs)	Reference	1.58 (1.20–2.11)
p value		0.001

Model 1: Adjusted for age, sex, smoking status, alcohol-drinking status, BMI and region

Model 2: Adjusted for age, sex, smoking status, alcohol-drinking status, BMI, region, WC, SBP, DBP, HbA1c, FBG, UA, exercise, sit, TC, TG, and LDL-C

BMI body mass index, *WC* waist circumference, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HbA1c* glycated hemoglobin A1c, *FBG* fasting blood glucose, *UA* uric acid, *TC* total cholesterol, *TG* triglyceride, *LDL-C* low-density lipoprotein cholesterol

$p=0.001$). The association between skipping breakfast and MASLD was consistent and robust across all models. After full adjustment, breakfast skipping remained independently associated with higher odds of MASLD.

Table 3 displays the logistic regression analysis examining the relationship between late dinner consumption and MASLD prevalence. In the crude analysis, participants with late dinner consumption demonstrated a significantly elevated odds ratio of 1.36 (95% CI 1.11–1.66, $p=0.002$) for MASLD compared to those without this eating pattern. Sequential adjustment models revealed consistent findings, with Model 1 (adjusted for basic demographic and lifestyle variables) showing an OR of 1.36 (95% CI 1.08–1.71, $p=0.009$), while the fully adjusted Model 2 maintained statistical significance with an OR of 1.37 (95% CI 1.03–1.82, $p=0.03$). These data provide evidence of a positive association between late dinner consumption and MASLD prevalence, demonstrating the robustness of this relationship even after comprehensive covariate adjustment.

Table 4 demonstrates a clear dose–response relationship between the severity of irregular eating behaviors and MASLD. In the fully adjusted model (Model 2), the odds of having MASLD were significantly higher for both the "Medium" irregular eating group (OR = 1.39, 95% CI 1.01–1.91, $p=0.045$) and the "Severe" irregular eating group (OR = 1.65, 95% CI 1.17–2.34, $p=0.004$), when compared to those with no irregular habits. The association for the "Severe" irregular eating group was particularly robust, remaining statistically significant across all analytical models, including unadjusted and partially adjusted analyses. This graded increase in the odds of MASLD with a greater number of irregular habits, persisting after comprehensive adjustment, underscores irregular eating patterns as an independent correlate of MASLD in this study population.

Subgroup analyses of the association between irregular eating behaviors and MASLD

The subgroup analysis in Fig. 2 reveals that the association between skipping breakfast and MASLD was significantly modulated by glucose status (*p* for interaction = 0.004). This interaction was driven by a strong and highly significant association among individuals with NGT, where skipping breakfast more than doubled the

Table 3 Logistic regression analysis of the association between late dinner consumption and MASLD

Logistic regression models	Categories of late dinner consumption	
	No	Yes
Unadjusted ORs (95%CIs)	Reference	1.36 (1.11–1.66)
p value		0.002
Model 1 ORs (95%CIs)	Reference	1.36 (1.08–1.71)
p value		0.009
Model 2 ORs (95%CIs)	Reference	1.37 (1.03–1.82)
p value		0.03

Model 1: Adjusted for age, sex, smoking status, alcohol-drinking status, BMI and region

Model 2: Adjusted for age, sex, smoking status, alcohol-drinking status, BMI, region, WC, SBP, DBP, HbA1c, FBG, UA, exercise, sit, TC, TG, and LDL-C

BMI body mass index, *WC* waist circumference, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HbA1c* glycated hemoglobin A1c, *FBG* fasting blood glucose, *UA* uric acid, *TC* total cholesterol, *TG* triglyceride, *LDL-C* low-density lipoprotein cholesterol

Table 4 Logistic regression analysis of the association between irregular eating behaviors and MASLD

Logistic regression models	Categories of irregular eating behaviors		
	No	Medium	Severe
Unadjusted ORs (95%CIs)	Reference	1.16 (0.92–1.46)	1.44 (1.13–1.83)
p value		0.19	0.003
Model 1 ORs (95%CIs)	Reference	1.19 (0.91–1.55)	1.53 (1.16–2.02)
p value		0.2	0.003
Model 2 ORs (95%CIs)	Reference	1.39(1.01–1.91)	1.65 (1.17–2.34)
p value		0.045	0.004

Model 1: Adjusted for age, sex, smoking status, alcohol-drinking status, BMI and region

Model 2: Adjusted for age, sex, smoking status, alcohol-drinking status, BMI, region, WC, SBP, DBP, HbA1c, FBG, UA, exercise, sit, TC, TG, and LDL-C

BMI body mass index, *WC* waist circumference, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HbA1c* glycated hemoglobin A1c, *FBG* fasting blood glucose, *UA* uric acid, *TC* total cholesterol, *TG* triglyceride, *LDL-C* low-density lipoprotein cholesterol

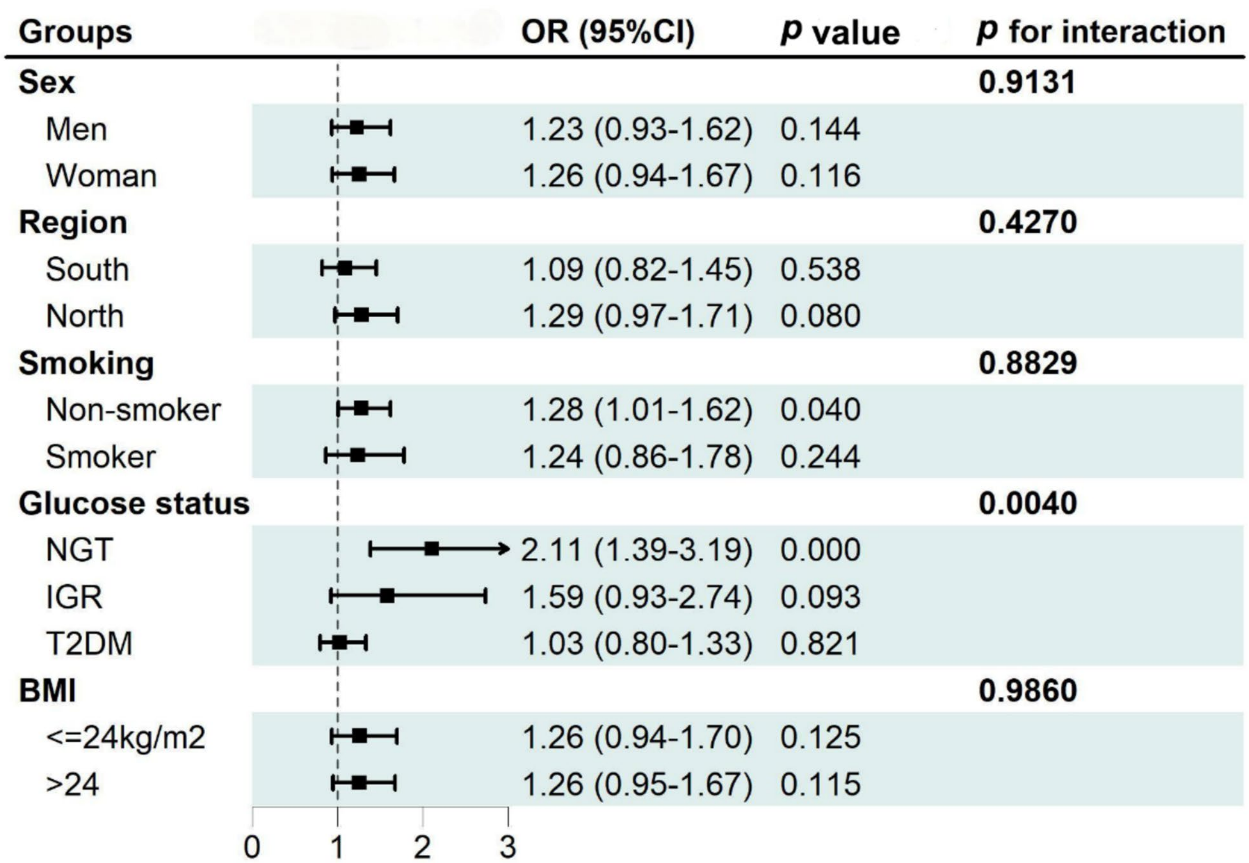

Fig. 2 Stratified odds ratios (ORs) with 95% confidence intervals (CIs) for MASLD based on skipping breakfast. Skipping breakfast as a categorical variable in the model. Adjusted for age, sex, smoking status, alcohol-drinking status, BMI, region, WC, SBP, DBP, HbA1c, FBG, UA, exercise, sit, TC, TG, and LDL-C. *BMI* body mass index, *WC* waist circumference, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HbA1c* glycated hemoglobin A1c, *FBG* fasting blood glucose, *UA* uric acid; *TC* total cholesterol, *TG* triglyceride, *LDL-C* low-density lipoprotein cholesterol

odds of having MASLD (OR = 2.11, 95% CI 1.39–3.19, $p < 0.001$). In contrast, this effect was attenuated and not statistically significant in those with IGR or T2DM. No significant associations or interaction effects were observed in the other subgroups stratified by sex, region, or BMI (all p for interaction > 0.05). These findings highlight a significant heterogeneity in the association between skipping breakfast and MASLD, emphasizing a particularly strong impact among individuals with normal glucose metabolism.

The subgroup analysis in Fig. 3 reveals that the association between late dinner consumption and MASLD is not uniform, but is significantly modulated by key factors, with regional differences being particularly prominent. A strong interaction with region was observed (p for interaction = 0.0180). Specifically, the association was robust and highly significant in the Northern population (OR = 1.59, 95% CI 1.20–2.11, $p = 0.001$), whereas it was entirely non-significant in the Southern population, indicating that geographic region is a key moderator of this association. Furthermore, glucose status also played a significant moderating role (p for interaction = 0.0241). The association was strongest in individuals with NGT (OR = 2.10, 95% CI 1.36–3.21, $p = 0.001$), but this effect diminished with worsening glucose metabolism and was no longer statistically significant in individuals with IGR or T2DM. Notably, although statistically significant associations were observed within certain specific subgroups, such as in men (OR = 1.44, $p = 0.012$), non-smokers (OR = 1.36, $p = 0.013$), and BMI groups, the corresponding p-values for interaction were not significant. This indicates that there is no fundamental statistical difference in the odds of MASLD associated with late dinner consumption between men and women, smokers and non-smokers, or across different BMI levels.

The subgroup analysis presented in Fig. 4 aimed to test for potential effect modification, but revealed no statistically significant interactions for any of the variables tested (all p for interaction > 0.05). This primary finding indicates that the association between irregular

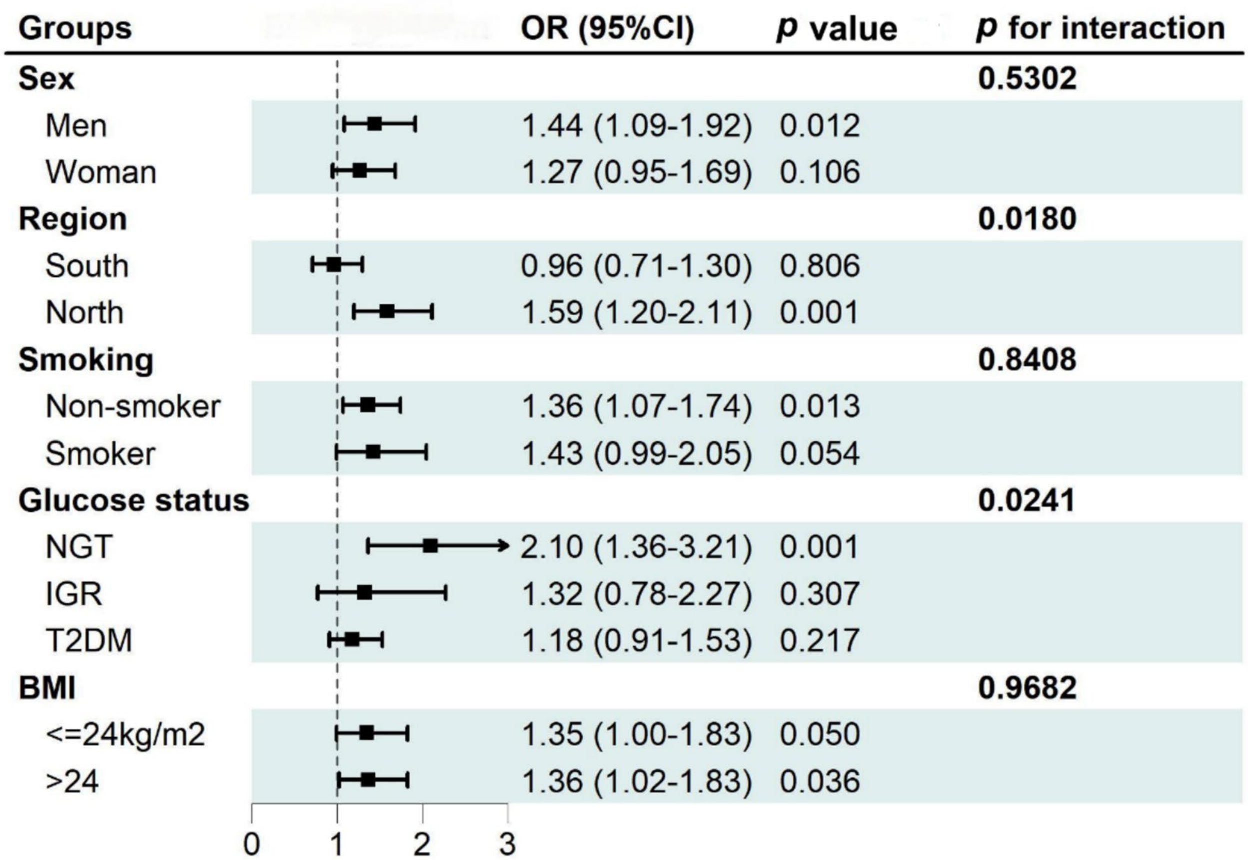

Fig. 3 Stratified odds ratios (ORs) with 95% confidence intervals (CIs) for MASLD based on late dinner consumption. Late dinner consumption as a categorical variable in the model. Adjusted for age, sex, smoking status, alcohol-drinking status, BMI, region, WC, SBP, DBP, HbA1c, FBG, UA, exercise, sit, TC, TG, and LDL-C. *BMI* body mass index, *WC* waist circumference, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HbA1c* glycated hemoglobin A1c, *FBG* fasting blood glucose, *UA* uric acid, *TC* total cholesterol, *TG* triglyceride, *LDL-C* low-density lipoprotein cholesterol

eating behaviors and MASLD is broadly consistent across demographic and clinical strata like sex, smoking status, glucose status, and BMI. While point estimates for the association appeared stronger in certain groups, such as those with NGT (OR = 1.62) or in the Northern region (OR = 1.28), the non-significant interaction tests confirm these apparent differences were not statistically significant. This general lack of interaction underscores that the adverse association between irregular eating behaviors and the prevalence of MASLD has broad applicability across diverse populations, though a noteworthy trend was still observed for geographic region.

Discussion

This multicenter cross-sectional study examined the association between skipping breakfast, late dinner consumption, and their combined effects on the prevalence of MASLD in Chinese adults. Our findings demonstrate that these behaviors are independently associated with higher odds of MASLD, after adjusting for key demographic, lifestyle, and metabolic factors. Notably, a significant dose–response relationship was observed, with the highest prevalence of MASLD found in individuals who both skipped breakfast and consumed late dinners. As the study population was recruited from specialized diabetes, obesity, and metabolic disease clinics, these findings are most directly applicable to the management of high-risk patients with existing metabolic disorders.

A key and novel finding of our study is that the link between irregular eating behaviors, particularly late dinner consumption, and MASLD differs significantly by geographic region. The association was strong and robust in the Northern population, yet absent in the Southern cohort. This heterogeneity may be explained by the profound differences in baseline dietary patterns between the two regions. The typical Northern diet, often higher in salt, saturated fats, and refined wheat, may place a greater baseline metabolic load on the liver [15, 17]. The addition of irregular eating could therefore act as a

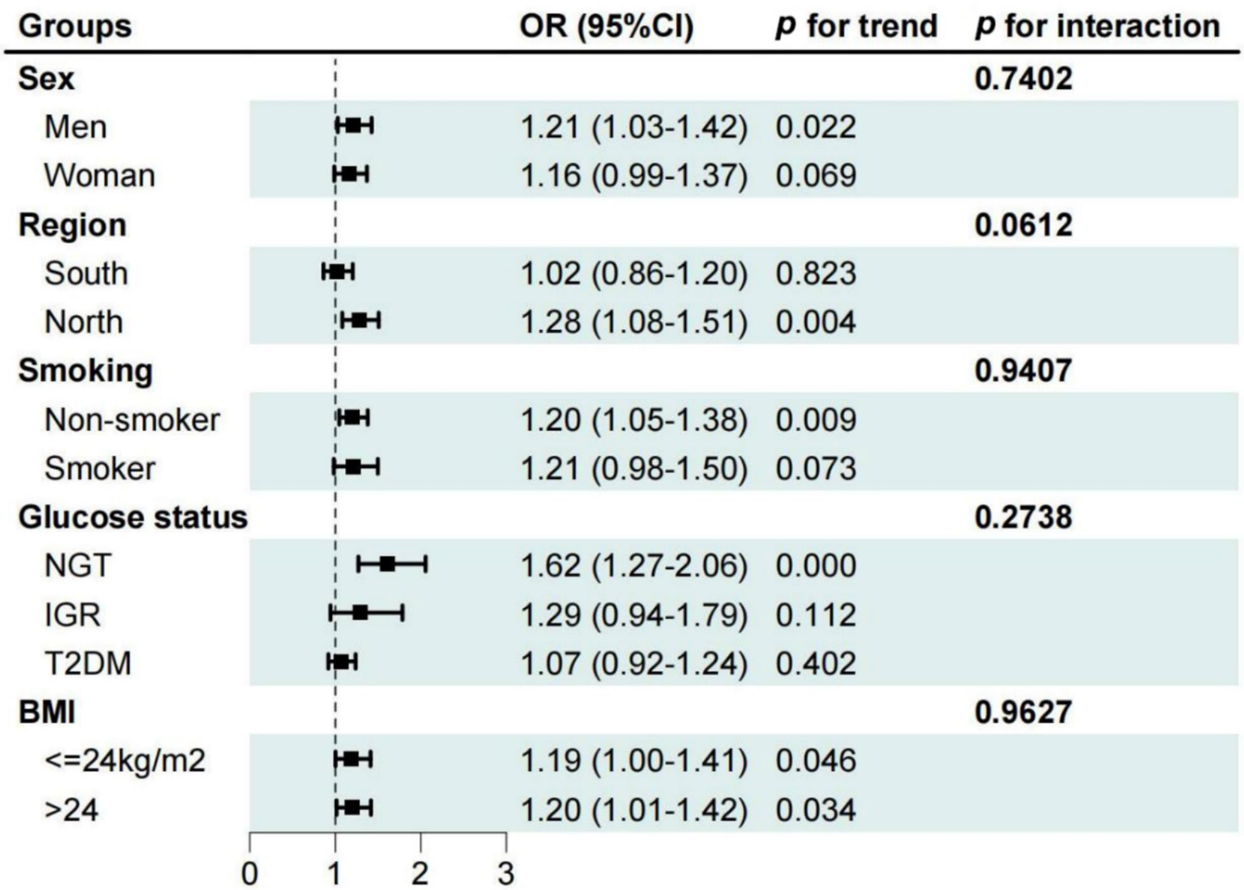

Fig. 4 Stratified odds ratios (ORs) with 95% confidence intervals (CIs) for MASLD based irregular eating behaviors. Irregular eating behaviors as a continuous variable in the model. Adjusted for age, sex, smoking status, alcohol-drinking status, BMI, region, WC, SBP, DBP, HbA1c, FBG, UA, exercise, sit, TC, TG, and LDL-C. *BMI* body mass index, *WC* waist circumference, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HbA1c* glycated hemoglobin A1c, *FBG* fasting blood glucose, *UA* uric acid, *TC* total cholesterol, *TG* triglyceride, *LDL-C* low-density lipoprotein cholesterol

"second hit", creating a synergistic effect that overwhelms hepatic metabolic capacity and promotes steatosis. Conversely, the traditional Southern diet, typically lighter and richer in rice, vegetables, and fish, may confer a degree of "metabolic resilience" [15, 17]. The higher concentration of omega-3 fatty acids from fish in Southern diet, in particular, might offer direct protection against liver steatosis, independent of its beneficial effects on other metabolic disorders [15, 18]. This healthier baseline diet could buffer the liver against the metabolic disruption caused by an irregular eating schedule, explaining the null association in the South. Beyond dietary compositions, these regional disparities may also be attributable to differences in genetic susceptibility, as a higher prevalence of NAFLD-related genetic polymorphisms has been observed in the Northern Han Chinese population [14, 19–21]. These findings underscore the importance of considering the background dietary matrix when evaluating the health effects of specific eating patterns.

Skipping breakfast has been associated with an elevated risk of being overweight and obesity [22]. It has been shown to lead to significant changes in metabolic parameters, including higher fasting blood glucose [23], triglycerides, and cholesterol levels [23–25], while HDL cholesterol is significantly reduced [23]. This disruption in lipid and glucose metabolism is thought to result from the combination of excessive lipid absorption and compensatory overeating later in the day [10, 26, 27]. The impact of skipping breakfast extends to circadian rhythm disruption, which could further impair metabolic functions [27, 28]. Skipping breakfast has been shown to exacerbate liver steatosis [23] and accelerate the progression of metabolic dysfunction-associated fatty liver disease (MAFLD), likely due to disruptions in circadian timing and differences in insulin sensitivity [29]. Additionally, skipping breakfast is linked to a higher risk of long-term cardiovascular mortality, especially in individuals having MAFLD, but not in those without such a condition [29].

Our study found that skipping breakfast is associated with the prevalence of MASLD.

Okada et al. discovered that Japanese women who had late dinners were more prone to skipping breakfast. Having a late dinner was associated with a greater probability of being overweight or obese [30]. Having an earlier evening meal led to beneficial alterations in weight loss and plasma cardiometabolic risk indicators during a weight loss program [31]. The late eating pattern might have an impact on circadian genes (SIRT1 and CLOCK loci), which could lead to late eaters being more likely to gain weight and having a lower capacity to lose it. Individuals with minor alleles at both the SIRT1 and CLOCK loci exhibited a significantly higher resistance to weight loss, which was associated with a preference for late evenings [32]. A delayed circadian rhythm in those who eat late is also related to the lower insulin sensitivity [33] and metabolic alterations [34] via hormonal changes, which results in being overweight and obesity [35]. Late dinner consumption, particularly when aligned close to sleep time, may further impair nocturnal metabolic processes, including insulin sensitivity and fat oxidation, thereby promoting hepatic fat accumulation [36–38]. Our study found that late dinner consumption is associated with the prevalence of MASLD.

Our results corroborate and extend previous research linking irregular eating behaviors, such as skipping breakfast and late dinner consumption, to adverse metabolic profiles. While prior studies have primarily focused on individual dietary habits, such as skipping breakfast [9, 10] or late-night dinner eating [39], our study uniquely examines the synergistic effect of these behaviors. This combined effect may stem from complex interactions among multiple underlying mechanisms. First, the combination amplifies the disruption of circadian rhythms. Circadian rhythms regulate the regulation of genes involved in triglyceride synthesis (e.g., Gpat2, Lipin1/2, Agpat1/2, Dgat2), fatty acid synthesis, β-oxidation, and influence the expression of key lipid reaction nuclear receptors (e.g., LXRs and PPARs) [40–42]. The role of the circadian machinery in lipid metabolism has been linked to its association with hepatic steatosis [43, 44]. Furthermore, circadian misalignment can promote inflammation and oxidative stress, further contributing to liver injury [45]. Late dinner consumption extends the eating window and increases the likelihood of subsequent breakfast skipping, creating a cycle that further exacerbates circadian disruption. In addition to the circadian component, adverse endocrine profiles are also likely involved. On one hand, Studies have shown that individuals who eat breakfast exhibit higher levels of satiety hormones, such as peptide YY (PYY) and glucagon-like peptide-1 (GLP-1), during the midday period, whereas those who skip breakfast do not experience such increase in hormone levels [46]. These hormones are related to satiety, and skipping breakfast may affect the secretion of these hormones, thereby influencing hunger and appetite control. On the other hand, late dinner consumption decreases leptin levels while increasing ghrelin levels. This hormonal imbalance makes individuals more likely to feel hungry after dinner, thereby increasing food intake, especially a heightened desire for high-calorie foods [13]. Indeed, during a 16-h wake period, late eating reduced average leptin levels by 16% and increased ghrelin levels by 34% [13].

A key question arising from our findings is whether the association between irregular eating behaviors and MASLD is driven by total caloric intake or by the metabolic consequences of the eating pattern itself. Although our study did not capture quantitative dietary data, which represents an important limitation, existing literature suggests several potential mechanisms. Research on caloric compensation following meal skipping yields mixed results, ranging from complete or over-compensation [47, 48], to incomplete compensation [49, 50]. Research indicates that individuals who skip breakfast typically fail to fully compensate for the missed calories in subsequent meals, resulting in a net caloric deficit of 200–400 kcal [49, 50]. Conversely, late-night dinner eating is associated with increased total caloric intake, particularly elevated nighttime caloric consumption [51]. Therefore, individuals exhibiting both irregular eating behaviors may experience what we term "temporal caloric misalignment"—characterized by relative caloric insufficiency during the day but excessive intake at night. This eating pattern not only disrupts normal metabolic circadian rhythms [52] but may also exacerbate hepatic fat accumulation through pathways involving insulin sensitivity, lipid metabolism, and inflammatory responses [13, 53]. Supporting this hypothesis, Bo et al.'s prospective cohort study demonstrated that consuming a greater proportion of daily caloric intake at dinner was associated with a 56% increased risk of NAFLD development, even after adjusting for total energy intake [51]. Furthermore, irregular meal patterns, breakfast skipping, and nighttime eating are all closely associated with adverse effects on liver health [52]. Collectively, the association between irregular eating patterns and MASLD severity in our study likely reflects the metabolic consequences of when calories are consumed rather than simply how many calories are consumed. This temporal misalignment of energy intake with circadian metabolism may explain why participants with both behaviors (breakfast skipping + late-night dinner eating) showed the highest disease severity, representing a "double metabolic hit" of circadian disruption plus evening caloric excess. Future studies should incorporate detailed temporally-resolved nutritional intake data to further elucidate the role of dietary timing patterns in MASLD pathogenesis.

To our knowledge, this is the largest nationwide multicenter study to investigate the association between irregular eating behaviors (skipping breakfast and late dinner consumption) and their combined effect on the prevalence of MASLD in a Chinese population. Strengths of this study include its multicenter design, diverse sample, and extensive data on lifestyle and metabolic factors. However, several limitations should be acknowledged. First, as a cross-sectional study, we cannot establish causality between irregular eating behaviors and MASLD. Second, our dietary assessment has inherent constraints. The data were self-reported, which are subject to recall bias, and our questionnaire focused on behavioral patterns rather than quantitative intake. Consequently, we were unable to assess the role of total caloric intake, or evaluate the consumption of specific dietary components such as processed foods and sugary drinks, which represent important potential confounders. Third, we did not collect detailed information regarding socioeconomic status (SES). This represents a significant limitation, as socioeconomic factors may serve as important confounders or mediators of our observed associations. Lower SES individuals may be more prone to breakfast skipping due to work schedule constraints, multiple jobs, or limited meal preparation time, and are more likely to engage in shift work leading to circadian rhythm disruption [54–57]. Additionally, socioeconomic factors are independently associated with non-alcoholic fatty liver disease risk and may influence food access and quality [58, 59]. Fourth, regarding dietary composition, irregular eating patterns may correlate with increased consumption of ultra-processed foods and sugary drinks, which are independently associated with MASLD risk [60]. Late eating episodes may particularly involve more processed, convenient foods rather than home-prepared meals, and evening consumption of high-fructose beverages may have particularly deleterious effects on hepatic metabolism [61–63]. Fifth, our study population was recruited exclusively from specialized diabetes, obesity, and metabolic disease clinics. This clinical sample has a much higher baseline risk for MASLD than the general population and therefore is not representative of the general community. Consequently, the generalizability of our findings to broader populations remains limited.

Despite these limitations, our findings carry important clinical and public health implications for several reasons. First, meal timing interventions are modifiable regardless of SES. Second, the metabolic effects of circadian disruption occur independently from food quality [64–66]. Healthcare professionals should counsel patients about the potential metabolic consequences of irregular eating behaviors and encourage them to adopt a consistent breakfast routine and avoid late-night meals. Public health interventions should promote healthy eating patterns and raise awareness about the importance of meal timing regularity. Future studies should incorporate comprehensive SES assessment, detailed dietary composition analysis, and stratified analyses to identify vulnerable populations. Moreover, longitudinal research is needed to establish causal relationships between irregular eating behaviors and MASLD, elucidate the underlying mechanisms, and evaluate the efficacy of interventions targeting dietary timing patterns for the prevention and management of MASLD. Nevertheless, it is important to recognize that temporal eating patterns may represent an independent risk factor worthy of clinical attention alongside dietary quality interventions.

Conclusions

In conclusion, this multicenter cross-sectional study demonstrates that irregular eating behaviors are significantly associated with a higher prevalence of MASLD. These findings highlight the importance of regular meal timing for metabolic health. Because the study population was recruited from specialized metabolic disease clinics, the implications of these findings are most directly relevant for managing high-risk patients. Future longitudinal and interventional studies in broader populations are needed to establish a causal relationship and confirm whether improving meal patterns is an effective strategy for preventing and managing MASLD.

Abbreviations

MASLD	Metabolic dysfunction-associated steatotic liver disease
BMI	Body mass index
NGT	Normal glucose tolerance
IGR	Impaired glucose regulation
T2DM	Type 2 diabetes mellitus
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
FBG	Fasting blood glucose
2hBG	2-Hour postprandial blood glucose
HbA1c	Glycated hemoglobin A1c
TC	Total cholesterol
TG	Triglyceride
HDL-C	High-density lipoprotein cholesterol
LDL-C	Low-density lipoprotein cholesterol
ALT	Alanine aminotransaminase
AST	Aspartate aminotransferase
GGT	Gamma-glutamyl transferase
UA	Uric acid
LFC	Liver fat content
ORs	Odds ratios
CIs	Confidence intervals
MAFLD	Metabolic dysfunction-associated fatty liver disease
PYY	Peptide YY
GLP-1	Glucagon-like peptide-1
SES	Socioeconomic status

Acknowledgements
We thank the study participants for all the works done for this study.

Author contributions
Hua Bian, Mingfeng Xia, and Xin Gao designed and supervised the research; Miao Zhang, Xiaoyang Sun, Lili Zheng, Yufang Bi, Qiang Li, Lirong Sun, Fusheng Di, Yushan Xu, Dalong Zhu, Yanyan Gao, Yuqian Bao, Yao Wang and Lanjie He conducted the research and collected data from centers across the country; Xingxing Ren conducted the statical analysis; Xingxing Ren drafted the manuscript draft; Jian Gao, Mingfeng Xia, and Hua Bian reviewed and edited the manuscript; Hua Bian had primary responsibility for final content. All named authors have reviewed and approved the final manuscript for submission.

Funding
This work was supported by the Noncommunicable Chronic Diseases-National Science and Technology Major Project (Grant No.2023ZD0508703 to Hua Bian), the National Natural Science Foundation of China (Grant No.82370870 to Hua Bian and Grant No.82000821 to Xingxing Ren), the Science and Technology Commission of Shanghai Municipality (Grant No.22Y31900302 to Hua Bian), the Shanghai Municipal Health Commission (Grant No. 202240295 to Hua Bian), and the Shanghai Sailing Program (20YF1406300 to Xingxing Ren).

Data availability
The data and materials used and/or analyzed during the current study are accessible upon reasonable request to the corresponding author.

Declarations

Ethics approval and consent to participate
The study protocol followed the guidelines of Research Ethics Committees of Zhongshan Hospital affiliated to Fudan University (approval number: 2011-6) and protocols in other involved hospitals were performed according to the guidelines of the Declaration of Helsinki, and approved by the Institutional Review Board.

Consent for publication
Not applicable.

Competing interests
The authors declare no conflict of interest.

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Received: 25 March 2025 Accepted: 15 July 2025
Published online: 02 August 2025

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