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Research Article

Associations between Eating Behaviors, Body Composition, and Mental Health in Asian and White Adults with Metabolic Dysfunction-associated Steatotic Liver Disease

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Background

Metabolic dysfunction-associated steatotic liver disease (MASLD) is affected by metabolic, psychological, and behavioral factors, with potential differences in pathophysiological pathways across racial groups.

Objective

This study aims to investigate the relationship between eating behaviors, emotional functioning, and anthropometric differences in Asian and maintain at various stages of liver disease.

Methods

A retrospective analysis was conducted on 98 MASLD patients (70 White and 28 Asian) from a Southern California liver clinic. Participants completed validated questionnaires assessing depressive and anxiety symptoms, eating behaviors, and quality of life. Body composition was measured using bioelectrical impedance analysis, and liver health was assessed through transient elastography. Multivariate regression and mediation analyses examined associations and pathways among psychological, behavioral, and clinical variables, with race as a moderator.

Results

White participants had higher body mass index (BMI) and body fat percentage, but liver steatosis and stiffness were similar between groups. In Asians, hepatic steatosis correlated most strongly with body fat mass, whereas in Whites, emotional eating was more closely linked to BMI and body fat percentage compared to Asians. Whites reported higher anxiety and lower quality of life. Emotional eating was associated with hepatic steatosis in both groups, but only in Whites did BMI and percent body fat mediate this relationship. Among Asians, emotional eating was linked to liver fat independent of body composition.

Conclusion

Distinct racial pathways exist in MASLD progression, with Asians potentially developing fatty liver without overt body fat changes. These findings highlight the need for tailored diagnostic and clinical strategies that consider metabolic, behavioral, and psychological differences across racial groups.

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1. INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most common chronic liver disease worldwide and a leading cause of liver-related morbidity and mortality.[1,2] MASLD affects approximately one-third of the global population. In addition to becoming the leading indication for liver transplantation, MASLD is strongly associated with an increased incidence of hepatocellular carcinoma.[3,4] MASLD encompasses a spectrum of liver-related disorders beginning with steatosis and then transitioning from steatohepatitis to fibrosis, and finally, cirrhosis, with the fibrosis stage being the strongest predictor of liver-related mortality.[2] Defined by the presence of steatosis in more than 5% of hepatocytes, MASLD is also closely linked to metabolic syndrome-associated conditions, such as obesity, type 2 diabetes mellitus, dyslipidemia, hypertension, and cardiovascular disease.[1]

Although the overall risk of liver-related mortality among patients with MASLD is low relative to the risk of cardiovascular complications and non-liver malignancies, most patients with MASLD are asymptomatic, making diagnosis and clinical monitoring challenging. Therefore, individuals with obesity, metabolic syndromes, type 2 diabetes mellitus, dyslipidemia, or elevated aminotransferase levels should be routinely screened for MASLD. Non-invasive tests are often used to assess liver fibrosis, although liver biopsy remains the "gold standard." Although weight loss is efficacious in improving hepatic steatosis and regression of fibrosis,[5] successful long-term management of MASLD requires a multidisciplinary approach to address liver-specific issues and cardiovascular risks,[1] as well as associated psychological and psychosocial factors.[6,7] Prior research suggests that racial and ethnic differences in anthropometrics influence MASLD development.[8]

1.1. MASLD AND MENTAL HEALTH

MASLD and mental health disorders have a bidirectional relationship due to shared pathophysiological pathways that impact processes, such as inflammation, oxidative stress, and metabolic dysfunction.[9] In addition, symptoms of depression (26.3%), anxiety (37.2%), and stress (51.4%) occur at higher rates among patients with MASLD compared to the general population.[7,10] Psychological factors are also associated with severe liver histological abnormalities[11] and cortisol dysregulation.[12] Finally, low perceived social support and significant liver fibrosis have been independently linked to poorer quality of life, mental health, and maladaptive coping strategies among adults with MASLD.[13]

1.2. MASLD AND DIET

Dietary habits and eating behaviors strongly impact MASLD. Studies have found a significant prevalence of binge eating disorders in patients with MASLD,[14] and patients with MASLD often exhibit dysfunctional eating behaviors, which may hinder treatment success.[15]

1.3. MASLD AND ETHNICITY

Ethnicity is a notable predisposing factor that influences MASLD prevalence and progression, with Hispanics showing the highest risk and African Americans demonstrating the lowest risk of development.[8] Despite lower obesity rates, Asians have a significant prevalence of MASLD due to differences in body composition, such as central adiposity, visceral fat (VF) distribution, and insulin resistance.[16] Genetic factors, such as the patatin-like phospholipase domain-containing protein 3 polymorphism, also play a crucial role in the development of MASLD among non-obese Asians.[17] Ethnic factors are further complicated by significant differences in body composition between Asian and non-Asian populations. Indeed, the body mass index (BMI) cutoff for obesity is lower in Asians (27 kg/m^2) than in non-Asians (30 kg/m^2).[16] Furthermore, Asians are more likely to develop lean MASLD, defined as MASLD occurring with a BMI <25 kg/m^2 for non-Asian patients and a BMI <23 kg/m^2 for Asian patients.[17]

Given the contribution of psychological and behavioral factors to the development of MASLD, it is essential to recognize MASLD as a condition influenced in part by cognitive–behavioral factors to foster lifestyle interventions and improve stress management techniques.[6,7] To date, there has been limited research on how psychological and behavioral factors affect the development of MASLD among different races, particularly given the genetic and metabolic differences previously noted between Asian and non-Asian populations.

Therefore, this study aims to investigate the relationship between eating behaviors, emotional functioning, and anthropometric differences among Asian and White adults at various stages of liver disease. We hypothesized that we would observe a statistically significant mediation effect among eating behaviors and emotional functioning on MASLD, mediated through anthropometrics, and that this mediation effect differs significantly between White and Asian adults. By exploring these factors, we sought to uncover novel insights into developing effective and targeted interventional strategies for patients with MASLD.

2. MATERIALS AND METHODS

2.1. ETHICAL CONSIDERATIONS

This study was approved by the Hoag Foundation Institutional Review Board (IRB), which waived the requirement for formal ethics approval due to its retrospective nature. Furthermore, as this study involved a retrospective analysis of deidentified patient data, the requirement for informed consent from patients was waived by the IRB. Study data were anonymized and accessed only by the study authors to protect confidentiality and ensure patient privacy.

2.2. STUDY OVERVIEW

This is a retrospective study involving 98 patients with MASLD who presented to the Liver Clinic at Hoag Memorial Hospital Presbyterian in Southern California, from June 2022 to May 2023. The Liver Clinic at Hoag Hospital is a non-academic, tertiary, community hospital-based clinic where patients with MASLD were identified based on increased liver echogenicity on ultrasound, which was then corroborated using vibration-controlled transient elastography (VCTE). At the beginning of their clinical visit, all MASLD patients completed a composite paper questionnaire that included sociodemographic items and a set of validated instruments assessing depressive and anxiety symptoms, eating behaviors, health-related quality of life,

and self-regulation of eating. These instruments included the Generalized Anxiety Disorder-7 (GAD-7),[18] the Patient Health Questionnaire-9 (PHQ-9),[19] the Three-Factor Eating Questionnaire-R18 (TFEQ-R18),[20] the 12-Item Short Form Health Survey (SF-12),[21] and the Self-Regulation of Eating Behavior Questionnaire (SREBQ).[22]

2.3. ASSESSMENT OF SYMPTOMS OF DEPRESSION AND ANXIETY

Symptoms of depression and anxiety were assessed using the PHQ-9,[19] a 9-item self-report inventory for the screening and severity of depressive symptoms, and the GAD-7,[18] a 7-item self-report inventory that screens for the severity of anxiety symptoms. Participants' scores on the PHQ-9 and GAD-7 were summed and assigned a level of symptom severity according to standard cutoff scores (PHQ-9: 0–4 = "Minimal" symptoms of depression, 5–9 = "Mild" symptoms, 10–14 = "Moderate" symptoms, 15–19 = "moderately severe" Symptoms, and 20–27 = "Severe" symptoms; GAD-7: 0–4 = "Minimal" symptoms of anxiety, 5–9 = "Mild" symptoms, 10–14 = "Moderate" symptoms, and 15 or more = "Severe" symptoms).

2.4. ASSESSMENT OF EATING BEHAVIORS

Eating behaviors of the study participants were assessed using the SREBQ, a measure of self-regulatory eating capacity,[22] and the TFEQR-18, a measure of three key dimensions of human eating behavior and a cornerstone in eating behavior research.[23]

The SREBQ begins with three screening questions (e.g., food products respondents find tempting, intentions not to eat tempting foods, and intentions to maintain a healthy diet) to exclude participants who lack healthy eating intentions.[22] Participants then answered the 5-item SREBQ using a 5-point Likert scale (e.g., "0 = Never" to "4 = Always"). Questions A, C, and E were then reverse-scored, and a scaled score was obtained by calculating the sum of all five questions (scores <2.8 = Low self-regulation, 2.8–3.5 = Moderate regulation, and >3.6 = High regulation of eating behaviors).[22]

Next, raw scores from each of the three TFEQ-R18 scales were converted to a percentage of the highest possible score, using Equation (1):

$$\%\ \text{of highest possible score} = \frac{S - L}{Rs} \times 100 \quad (1)$$

Where S = Raw score, L = Lowest possible raw score, and Rs = Range of possible scores (e.g., 24 for cognitive restraint, 36 for uncontrolled eating, and 12 for emotional eating).[20]

2.5. ASSESSMENT OF HEALTH AND LIFESTYLE FACTORS

The SF-12 was used to assess general health and lifestyle factors. This self-report measure discerns the impact of a participant's health and perceived quality of life on everyday functioning across four different domains (e.g., general mental health [psychological distress and well-being], limitations in usual role activities because of emotional problems, limitations in social activities due to physical health problems, and limitations in physical activities).[21] Participant scores on the SF-12 were evaluated using a four-step process, including "cleaning" out-of-range values and reverse scoring four items (1, 8, 9, and 10).[21] Indicator variables were then identified (0 = Not endorsed; 1 = Endorsed) for each item-response choice category and were then weighted (using regression coefficients from the general United States [US] population) and aggregated by adding a constant (regression intercept). Aggregate scores were then standardized.[21]

Liver stiffness was determined using VCTE with M probe and XL probe (FibroScan 502 Touch model, Echosens, France) by an experienced VCTE-certified technician blinded to clinical data. Patients fasted for at least 4 h before the examination. The procedure was performed in the supine position with the right arm adducted while holding the breath for 10 s. All patients were first scanned with the M probe (3.5 MHz) over the right liver lobe. If indicated by the machine, patients were re-evaluated using the XL probe (2.5 MHz). Ten measurements were made, and the interquartile range (IQR) was <30%. Test failure was defined when no stiffness measurement was obtained or when there were unreliable measurements (success rate <60% or IQR/median >30%).[24] Simultaneous liver steatosis measurements were obtained using controlled attenuation parameter (CAP).

Following the VCTE examination, the patient's height was measured. The patient was asked to remove their shoes and socks and step onto the scale (S-MFBIA InBody 970, InBody Company Limited, South Korea). Voice instructions were generated by the InBody device, and a medical assistant was present to ensure compliance. Briefly, the patient grasped the handles with their palms and thumbs, making contact with the electrodes. The patient stood motionless for about 1 min. Data from the device included body composition measurements, such as BMI, percent body fat (PBF), skeletal muscle mass (SMM), body fat mass (BFM), VF, and abdominal fat. All measurements were automatically sent to a printer.[25]

Patients were then evaluated by a hepatologist, including a complete medical history and physical examination. Laboratory studies, including a comprehensive metabolic panel, hemoglobin A1c, and lipid panel, were also obtained. All patients were evaluated for chronic viral hepatitis (anti-hepatitis C virus and hepatitis B surface antigen), Wilson disease (ceruloplasmin level), hemochromatosis (iron studies and hemochromatosis gene testing), alpha-1 anti-trypsin deficiency (A-1AT phenotype), and autoimmune disease (anti-nuclear antibody, anti-smooth muscle antibody, anti-mitochondrial antibody, immunoglobulin M, and immunoglobulin G). Patients with other chronic liver diseases or who drank more than three units of alcohol per week were excluded from this study. FibroScan and body composition analysis were performed on the same clinic day; laboratory studies were obtained within 3 months of the clinic visit.

2.6. STATISTICAL METHODS

Data distribution was examined using histograms. Most measurements, such as CAP, followed a normal distribution; however, some exhibited deviations from normality (e.g., median liver stiffness was right-skewed). Consequently, the Wilcoxon-rank-based transformation was employed as a sensitivity analysis to model ranking positions rather than the raw values. To obtain adjusted correlations, we initially regressed out age, gender, and education level as covariates to control for the confounding effects of our model, then utilized the residuals for either Pearson or Spearman

correlation, depending on data normality. For regression models, residual plots were used to assess model integrity. Independent *t*-tests or Wilcoxon rank-sum tests were employed to determine differences between races, depending on data normality. Mediation and moderated mediation analyses were conducted using the PROCESS macro for SAS (Models 4, 7, and 14) (version 9.4, SAS Institute Inc., US).[26] The mediators, including BMI, PBF, VF area, and BFM, all demonstrated reasonable normality. The psycho-behavioral measurements exhibited poor normality but served as predictors and should not affect the normality assumption driven by the outcome and residual. The 95% confidence interval (CI) of the mediator effect was estimated using 1000 bootstrapping samples. Race was tested as a moderator of the mediation pathways for both the eating behavior to the mediator (e.g., BMI; Model 7) and mediator to CAP (Model 14).[27]

3. RESULTS

3.1. PARTICIPANT CHARACTERISTICS

The study sample (n = 98) was selected and broadly reflects the ethnic distribution of the local community. Demographic characteristics are outlined in Table 1. The mean age was 58.3 ± 13.9 years, with 71.4% (n = 70) of individuals identifying as White and the remaining participants identifying as Asian (n = 28). The majority were female (68.4%), with a mean household size of 1.6 persons. Comprehensive body composition analysis shows a mean weight of 86.6 ± 4.3 kg, a mean BMI score of 31.2 ± 5.5, a mean BFM score of 33.9 ± 11.9 kg, a mean VF area score of 169.0 ± 56.3 cm^2, and a mean PBF score of 39.5 ± 7.2%.

3.2. BODY COMPOSITION AND LIVER HEALTH BY RACE

A univariate analysis examining body composition, liver stiffness, and steatosis by race was conducted. Findings (Table 2) suggested White participants (mean: 92.5 ± 17.0 kg, median: 93 kg, IQR: 80.7–103.4) weighed significantly more than Asian participants (mean: 72.6 ± 11.1 kg, median: 71.3 kg, IQR: 63.5–80.1 kg; $p \leqslant 0.01$) and had a higher BMI (mean: 32.6 ± 5.4, median, 33.1, IQR: 28.9–35.3) than Asian (mean: 27.7 ± 4.2, median: 26.7, IQR: 24.8–30.5; $p \leqslant 0.01$). White participants also had significantly higher BFM (mean: 38.5 ± 12.2 kg, median: 40.1 kg, IQR: 30.3–45.8 kg) than Asian participants (mean: 26.0 ± 6.1 kg, median: 25.1 kg, IQR: 22.2–27.8; $p \leqslant 0.01$). Similarly, SMM in White participants (mean: 30.7 ± 7.1 kg, median: 29.9 kg, IQR: 25.0–38.3 kg)

Table 1. Demographic distribution by race

Demographics	White (n=70)	Asian (n=28)	Total (n=98)	p
Age (years), mean±SD	58.3±14.25	58.3±13.37	58.3±13.93	0.94[a]
Gender (%)				
Female	46 (65.71)	21 (75.00)	67 (68.37)	0.37[b]
Male	24 (34.29)	7 (25.00)	31 (31.63)	
Education level[c] ()				
High school graduate	5 (7.25)	1 (3.57)	6 (6.19)	0.53[b]
College	24 (34.78)	6 (21.43)	30 (30.93)	
Associate's degree	9 (13.04)	2 (7.14)	11 (11.34)	
Bachelor's degree	16 (23.19)	10 (35.71)	26 (26.80)	
Post-baccalaureate	3 (4.35)	1 (3.57)	4 (4.12)	
Master's degree	9 (13.04)	7 (25.00)	16 (16.49)	
Doctorate degree	3 (4.35)	1 (3.57)	4 (4.12)	
Ethnicity				
Not Hispanic, Latino, or of Spanish origin	57 (85.07)	26 (100.00)	83 (89.25)	0.04[b]
Hispanic, Latino, or of Spanish origin	10 (14.93)	0 (0.00)	10 (10.75)	

Note: Data are expressed as n (%) unless otherwise stated. [a]Wilcoxon rank-sum test. [b]Chi-Square test [c]Fewer participants answered the related questions in the questionnaire. Abbreviation: SD: Standard deviation.

Table 2. Univariate analysis of body composition and liver health by race

Body composition	White (n, mean±SD, median, IQR)	Asian (n, mean±SD, median, IQR)	p
Weight (kg)	70, 92.48±16.96, 92.99, 80.94–103.42	28, 72.75±11.09, 71.33, 63.50–80.06	<0.01[b]
SMM (kg)	37, 30.69±7.10, 29.90, 24.99–38.33	22, 23.76±4.27, 22.20, 21.32–24.4	<0.01[a]
BFM (kg)	38, 38.51±12.14, 41.03 , 30.30–45.81	22, 26.03±6.14, 25.08 , 22.18–27.80	<0.01[b]
BMI (kg/m^2)	67, 32.59±5.35, 33.10, 28.90–35.31	27, 27.68±4.21, 26.66, 24.80–30.50	<0.01[b]
PBF (%)	38, 40.73±7.84, 41.25, 34.30–47.60	22, 37.29±5.51, 36.50, 34.60–40.70	0.08[b]
VFA (cm^2)	38, 190.58±54.63, 198.45, 148.90–226.60	21, 129.98±34.37, 133.9, 108.00–139.50	<0.01[b]
CAP (dB/m)	58, 293.38±57.44, 296.50, 259.00–334.00	26, 298.12±47.60, 306.00, 281.00–327.00	0.71[b]
MLS (k/Pa)	58, 9.53±10.43, 5.80, 4.30–9.5	27, 5.92±2.22, 5.50, 4.30–6.40	0.032[a]

Notes: [a]Wilcoxon rank-sum test; [b]Student t-test.
Abbreviations: BFM: Body fat mass; BMI: Body mass index; CAP: Controlled attenuation parameter; IQR: Interquartile range; MLS: Median liver stiffness; PBF: Percent body fat; SD: Standard deviation; SMM: Skeletal muscle mass; VFA: Visceral fat area.

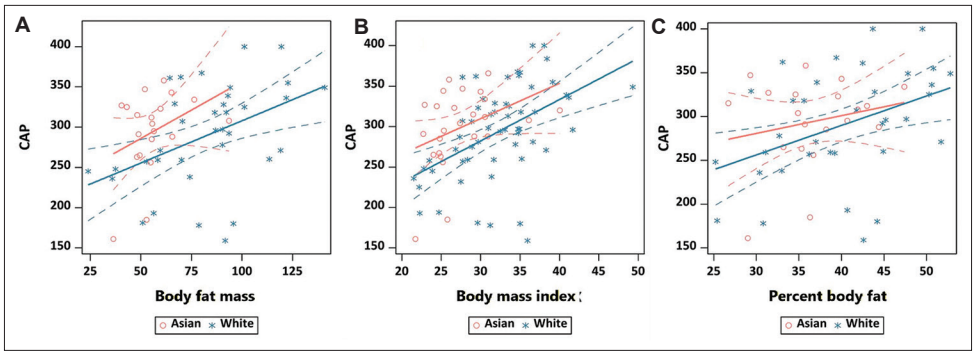

Figure 1. (A-C) Correlation between body composition and fatty liver by race
Notes: The Y-axis shows the residual value of the controlled attenuation parameter (CAP) after regressing out age, gender, and education levels. Solid lines represented the model-estimated value (slope), and dashed lines presented the 95% confidence interval of the predicted value at each given value on the X-axis.

was significantly higher than in Asian participants (mean: 23.8 ± 4.3 kg, median: 22.2 kg, IQR: 21.3–24.4 kg; $p \leqslant 0.01$). VF in White participants (mean: 86.5 ± 24.8 kg, median: 90.0 kg, IQR: 67.5–102.8 kg) was also significantly greater than Asian participants (mean: 59.0 ± 15.6 kg, median: 60.7 kg, IQR: 49.0–63.3 kg; $p \leqslant 0.01$). No significant differences were found in PBF ($p=0.06$), liver steatosis ($p=0.71$), and median liver stiffness ($p=0.32$) between White and Asian participants.

3.3. CORRELATION BETWEEN BODY COMPOSITION AND FATTY LIVER BY RACE

A correlation between body composition (e.g., BMI, BFM, and PBF) and hepatic steatosis (CAP) by race was also explored (Figure 1). BFM, BMI, and PBF were highly correlated with CAP scores among White and Asian participants. While the correlation appears consistent across all three anthropometric measurements among White participants, it varies among Asians, with BFM showing the strongest correlation, followed by BMI, and PBF showing the weakest correlation. The correlation between BMI and PBF by race showed no difference between White and Asian participants (Figure 2). The correlation pattern remained the same when the Wilcoxon ranking score was used instead.

3.4. PSYCHOLOGICAL, LIFESTYLE, AND EATING BEHAVIOR DIFFERENCES BY RACE

A univariate analysis was conducted to examine differences in psychological symptoms, lifestyle factors, and eating behaviors in White and Asian participants (Table 3). White participants (GAD-7 mean: 3.99 ± 4.39, median: 2, IQR: 0–6, $p \leqslant 0.01$) had a significantly higher level of anxiety compared to Asian participants (GAD-7 mean: 1.79 ± 3.44, median: 0, IQR: 0–2.5). White participants (SR-12 mean: 32.46 ± 6.84, median: 33.5, IQR: 28–38) also reported a significantly lower quality of life than their Asian counterparts (SR-12 mean: 35.75 ± 6.6, median: 37.5, IQR: 33.5–41.0, $p \leqslant 0.01$).

Most participants endorsed a minimal level of depression (n = 72, 73.5%); however, no significant difference in depression severity was found between White and Asian participants $p=0.06$. Similarly, no significant differences were found among White and Asian participants on any of the three TFEQ-R18 eating behavior factors, including uncontrolled eating ($p=0.11$), emotional eating ($p=0.35$), and self-regulation of eating ($p=0.72$). The correlation pattern remained the same when the Wilcoxon ranking score was used instead.

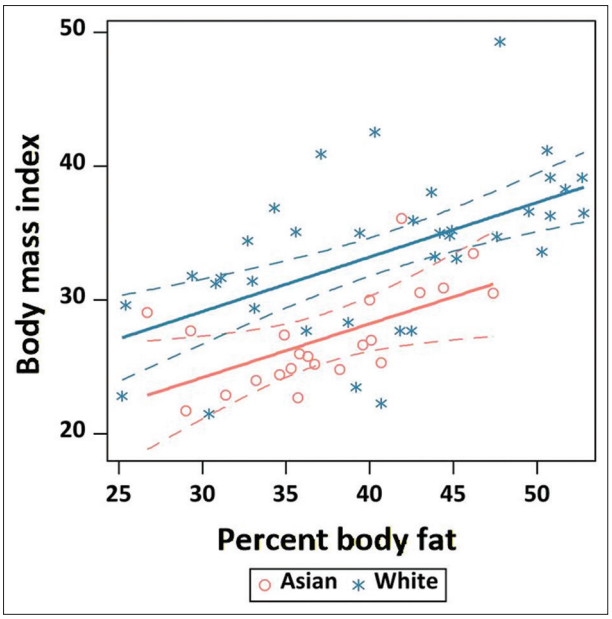

Figure 2. Correlation between body mass index and percent body fat by race
Notes: The Y-axis shows the residual value of body mass index after regressing out age, gender, and education levels. Solid lines represented the model-estimated value (slope), and dashed lines presented the 95% confidence interval of the predicted value at each given value on the X-axis.

3.5. ASSOCIATION BETWEEN EATING BEHAVIORS AND FATTY LIVER BY RACE

A similar association was found (Figure 3) between the TFEQ-R18 emotional eating factor scale and CAP scores among White (r = 0.35, 95% CI: 0.11–0.59) and Asian participants (r = 0.2, 95% CI: −0.23–0.64). Likewise, an association was found between the TFEQ-R18 factor scale for uncontrolled eating and the CAP scores of White (r = 0.24, 95% CI: 0.01–0.47) and Asian participants (r = 0.18, 95% CI: −0.25–0.60). Conversely, no associations were found between eating behaviors and liver stiffness among or across races.

Table 3. Univariate analysis of psychological symptoms, lifestyle factors, and eating behaviors by race

Questionnaire/Scale	White (*n*, mean±SD, median, IQR)	Asian (*n*, mean±SD, median, IQR)	*p*
GAD_7	70, 3.99±4.39, 2.00, 0–6	28, 1.79±3.44, 0, 0–2.50	<0.01[a]
PHQ_9	70, 4.43±5.34, 2.00, 0–9	28, 2.39±3.50, 1, 0–3	0.06[a]
T18_UES	70, 15.89±4.87, 16.00, 12–19	28, 14.25±3.53, 14, 11–17	0.11[b]
T18_EES	70, 5.66±2.46, 5.50, 3–7	28, 5.11±2.01, 5, 3–6.50	0.35[a]
SF12	70, 32.46±6.84, 33.50, 28–38	28, 35.75±6.60, 37.50, 33.50–41	0.01[a]
SREBQ	68, 2.81±0.49, 2.80, 2.60–3.10	27, 2.87±0.46, 3, 2.60–3.20	0.72[a]

Note: [a]Wilcoxon rank-sum test; [b]Student t-test.
Abbreviations: GAD_7: Generalized Anxiety Disorder questionnaire, Version 7; IQR: Interquartile range; PHQ_9: Patient Health Questionnaire, Version 9; SD: Standard deviation; SR12: 12-Item Short Form Health Survey; SREBQ: Self-Regulation of Eating Behavior Questionnaire; T18_EES: Three-Factor Eating Questionnaire, Revised 18, Emotional Eating Scale; T18_UES: Three-Factor Eating Questionnaire, Revised 18, Uncontrolled Eating Scale.

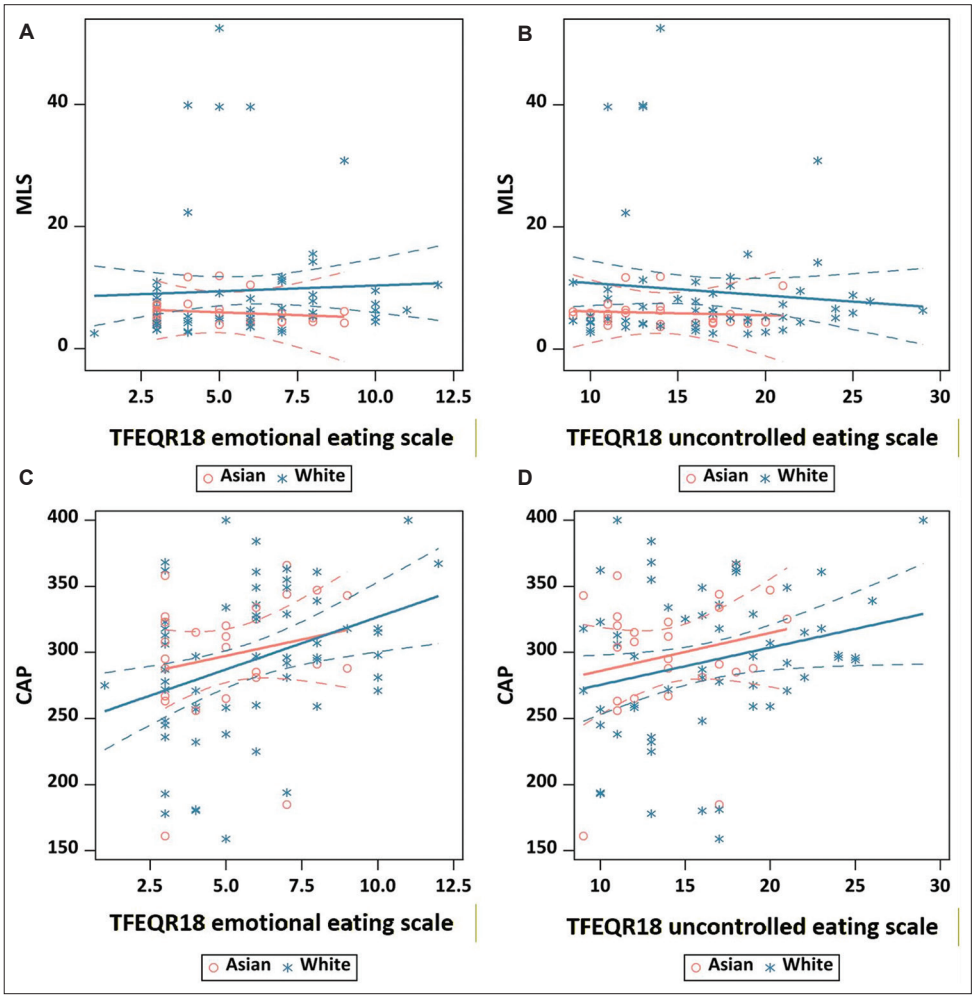

Figure 3. Association between eating behaviors and fatty liver by race. (A) Emotional eating and MLS. (B) Uncontrolled eating and MLS. (C) Emotional eating and CAP. (D) Uncontrolled eating and CAP.
Notes: The Y-axis shows the residual value of MLS or CAP after regressing out age, gender, and education levels. Solid lines represented the model-estimated value (slope), and dashed lines presented the 95% confidence interval of the predicted value at each given value on the X-axis.
Abbreviations: CAP: Controlled attenuation parameter; MLS: Median liver stiffness.

3.6. ASSOCIATION BETWEEN EATING BEHAVIORS AND BODY FAT BY RACE

While findings demonstrated racial differences in the relationship between eating behaviors (e.g., emotional eating and uncontrolled eating) and body fat (e.g., BMI, BFM, and PBF), none of the associations were statistically significant (Figure 4), most likely due to the study's small sample size. Among White participants, emotional eating was associated with BMI (emotional eating score [EES]: r = 0.34, 95% CI: 0.09–0.58), PBF (EES: r = 0.33, 95% CI: 0.03–0.62), and BFM (EES: r = 0.34, 95% CI: 0.02–0.65). However, for uncontrolled eating in White participants, it was only associated with BMI (uncontrolled eating scale [UES]: r = 0.35, 95% CI: 0.11–0.58) but not PBF (UES: r = 0.09, 95% CI: −0.20–0.39) and BFM (UES: r = 0.15, 95% CI: −0.16–0.47). As White

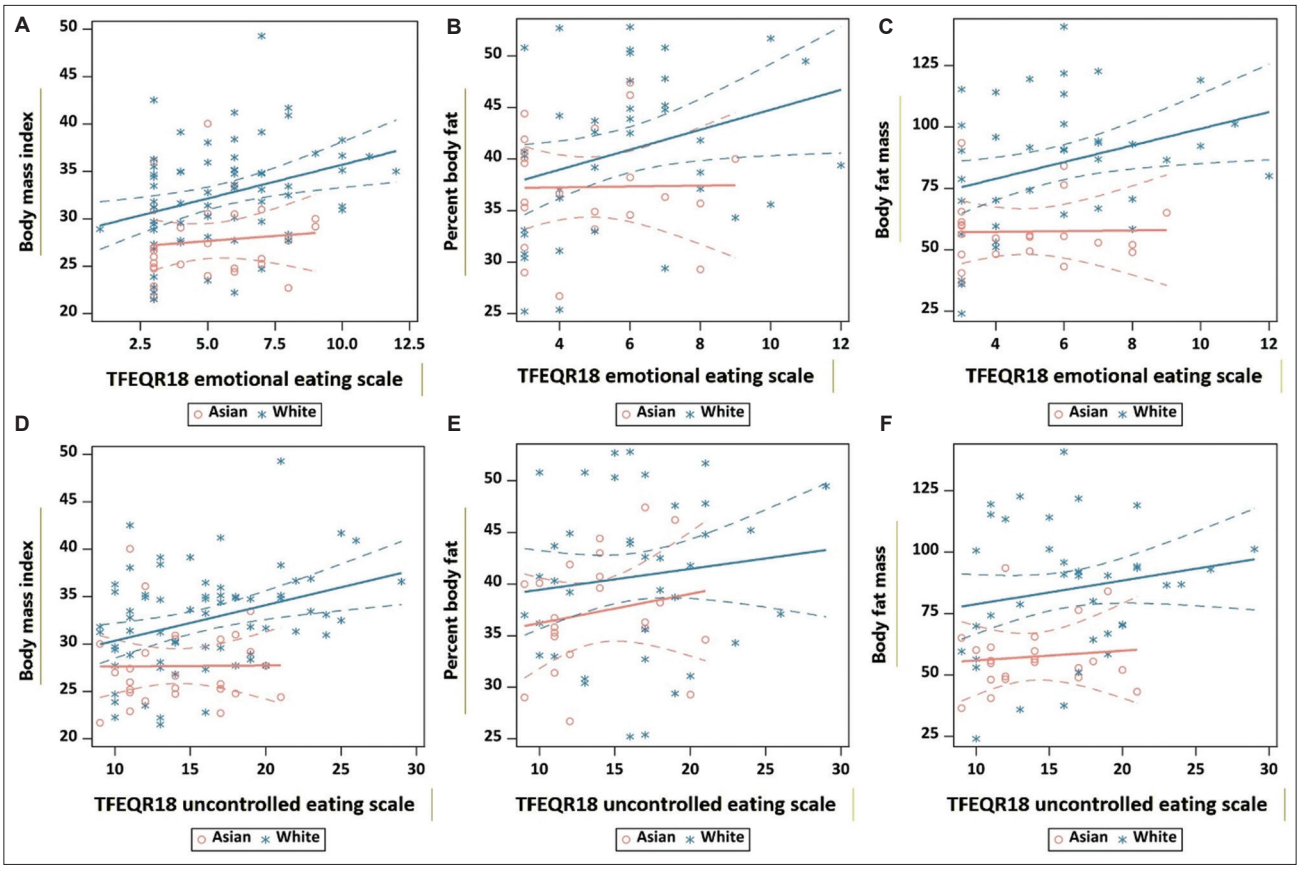

Figure 4. Association between eating behaviors and body fat by race. (A) Emotional eating versus BMI. (B) Emotional eating versus percent body fat. (C) Emotional eating versus body fat mass. (D) Uncontrolled eating versus BMI. (E) Uncontrolled eating versus percent body fat. (F) Uncontrolled eating versus body fat mass.
Notes: The Y-axis shows the residual value of body mass index, percent body fat, or body fat mass after regressing out age, gender, and education levels. Solid lines represented the model-estimated value (slope), and dashed lines presented the 95% confidence interval of the predicted value at each given value on the X-axis.

participants' TFEQ-R18 scores increased, they were more likely to have elevated BMI, PBF, and BFM scores. In contrast, there was little to no association among Asian participants between eating behaviors and BMI (UES: r = −0.05, 95% CI: −0.52–0.43; EES: r = 0.09, 95% CI: −0.04–0.59), PBF (UES: r = 0.18, 95% CI: −0.38–0.74; EES: r = 0.1, 95% CI: −0.46–0.65), or BFM (UES: r = 0.12, 95% CI: −0.7–0.94; EES: r = 0.05, 95% CI: −0.78–0.87). The correlation pattern remained the same when the Wilcoxon ranking score was used instead.

3.7. MEDIATOR AND MODERATED MEDIATION ANALYSIS

A mediation model analysis (Figure 5) was performed to determine whether body fat (M) mediated the relationship between emotional eating (X) and a participant's CAP score (Y). Figure 5 also shows additional mediation models with BMI, PBF, and BFM as candidate mediators for the association between EES and CAP scores. Results show that emotional eating positively predicts CAP scores (0.21, 95% CI: 0.00–0.41). This total effect was reduced to 0.11 (95% CI: −0.10–0.31) after controlling for the direct effect of BMI, indicating that BMI partially mediated the relationship between emotional eating and CAP scores with a mediator effect (indirect effect) of 0.10 (95% CI: 0.02–0.20). Similarly, the direct impact of emotional eating on CAP scores was reduced when controlling for PBF, indicating that PBF partially mediates this relationship.

A moderated mediation analysis (Figure 6) was also conducted to determine whether race (White vs. Asian) moderated the effect of body fat (M), which was previously identified as a mediator in the pathway between emotional eating (X) and CAP score (Y). The findings indicated that only the BMI (−0.13, 95% CI: −0.3–0.0) pathway was statistically significantly moderated by race, specifically in the path from emotional eating to body fat, but not in the pathway from body fat to CAP. PBF and BFM also demonstrated a trend toward a moderator effect but did not achieve statistical significance, with moderator effects of −0.19 (95% CI: −0.48–0.02) and −0.14 (95% CI: −0.42–0.03), respectively. Furthermore, statistically significant indirect effects were observed exclusively among White participants for BMI (0.14, 95% CI: 0.04–0.27) and PBF (0.19, 95% CI: 0.02–0.43) pathways. These indirect effects were not evident among Asian participants (BMI: −0.13, 95% CI: −0.3–0.0; PBF: −0.19, 95% CI: −0.48–−0.02).

Finally, a second moderated mediation analysis was conducted to determine whether race (White vs. Asian) moderated the mediation between body fat (M) and the hepatic steatosis CAP score (Y). Results indicated race did not appear to be a significant moderator in this pathway among any of the candidate mediators (BMI: 0.01, 95% CI: −0.14–0.29; BFM: 0.17, 95% CI: −0.22–0.77; and PBF: 0.01, 95% CI: −0.25–0.30).

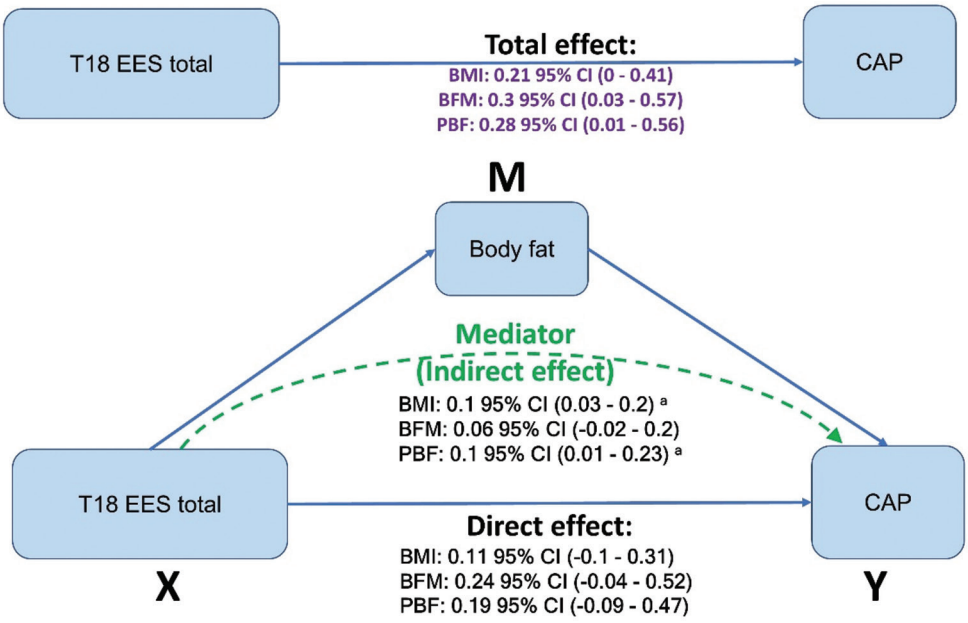

Figure 5. Mediation analysis
Notes: The total effect = Direct effect + Indirect effect. The indirect effect represents the mediation effect. [a]Indicates the statistical significance (95% CI, not including 0) for the individual mediator effect.
Abbreviations: BFM: Body fat mass; BMI: Body mass index; CAP: Controlled attenuation parameter; CI: Confidence interval; PBF: Percent body fat; T18 EES: TFEQR-18 emotional eating scale.

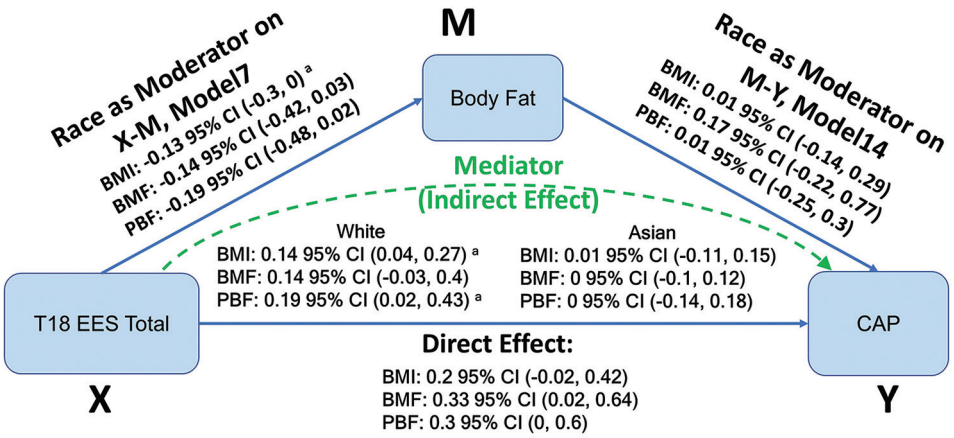

Figure 6. Mediated moderator analysis
Notes: The moderated mediator effect = Indirect effect (Asian) − Indirect effect (White). [a]Indicates the statistical significance (95% CI, not including 0) for either the moderated mediator effect or the individual mediator effect.
Abbreviations: BFM: Body fat mass; BMI: Body mass index; CAP: Controlled attenuation parameter; CI: Confidence interval; PBF: Percent body fat; T18 EES: TFEQR-18 emotional eating scale.

4. DISCUSSION

This study aimed to understand the relationship between food behaviors, emotional functioning, and anthropometric differences in Asian and White adults at various stages of liver disease. Three key findings were highlighted in this study. First, while White participants generally exhibited higher anthropometric measurements (e.g., body weight, BMI, fat mass, muscle mass, and VF) than Asians, liver health was comparable between the two groups. However, the relationship between body composition and liver fat differed significantly between the two races, with Asians demonstrating a stronger association between hepatic steatosis and BFM but a weaker correlation with BMI and body fat percentage. These findings are consistent with prior research demonstrating notable differences in body composition across racial cohorts. Specifically, prior studies reported that among White and Asian participants with similar BMI, Asians had a higher body fat percentage, greater abdominal obesity, higher intramyocellular lipid, and higher liver fat content than Whites.[28] Another study also reported that, for similar BMI, Asian participants' body fat percentage was 3–5% higher than that of Whites.[27] Similarly, when body fat percentage was held constant, BMI was consistently lower by 3–4 kg/m^2 among Asians than Caucasians.[27] As such, the findings of this study support previous research that overall body fat (e.g., absolute fat mass) among Asians is a more accurate indicator of health risks.[28]

The second notable finding suggests a nuanced relationship between psychological functioning and MASLD across

races. White participants with MASLD were found to have a slightly higher prevalence of anxiety on average compared to Asian participants. In addition, White participants with MASLD reported a lower quality of life, suggesting that they may experience a more significant impact from health-related issues than Asian participants. Previous research has indicated that anxiety is prevalent among patients with MASLD, with over one-third experiencing severe symptoms.[29] Interestingly, one prior study found that Asian Americans living with a chronic illness experience a higher burden of mental health issues, including depression and psychological distress.[30] These findings underscore the importance of considering cultural contexts and ethnicity when assessing anxiety and quality of life in diverse patient populations.[31]

The severity of depression was not significantly different between White and Asian participants. Although some inconsistencies exist in the literature, these results generally contradict previous findings. Depression and obesity have previously been demonstrated to have a bidirectional relationship, with each condition increasing the likelihood of developing the other.[32,33] Various biopsychosocial factors, including educational attainment, body image, binge eating, and physical health, are associated with the obesity–depression link.[34] Although the relationship between obesity and depression is well established, some studies have yielded contradictory results, emphasizing the necessity for further research to fully elucidate the underlying mechanisms.[35]

While dysfunctional eating behaviors did not appear to differ significantly between White and Asian participants, both emotional eating and uncontrolled eating were associated with higher levels of liver fat among the two groups. Racial differences were also observed in the relationship between dysfunctional eating behaviors (e.g., emotional eating and uncontrolled eating) and body fat levels (e.g., BFM and PBF); however, none of these associations were statistically significant. Specifically, White participants were more likely to exhibit elevated body fat levels when dysfunctional eating behaviors were present. Asian participants, however, demonstrated little to no association between eating behaviors and elevated body fat levels, suggesting that body fat levels in Asian participants may be less sensitive to variations in eating behavior compared to White participants. Previous research on eating disorders and body image in different ethnic groups has revealed complex and sometimes contradictory patterns.[36-38] While some studies reported that White women demonstrate higher levels of disordered eating, dieting behaviors, and body dissatisfaction than other ethnic minorities,[36,37] other studies observed similar eating disorder psychopathology across ethnicities.[38]

The final significant finding of this study was the mediation effect of body composition across races on liver steatosis. The results indicated that emotional eating was associated with higher liver steatosis levels. However, when BMI and PBF were considered, the direct impact of emotional eating on hepatic steatosis decreased, suggesting that BMI and PBF partially explain the relationship between emotional eating and fatty liver. Among White participants, BMI and PBF were still found to play a mediating role in the relationship between emotional eating and the degree of hepatic steatosis. This suggests that, among Asians, emotional eating may directly affect hepatic steatosis levels, without an intermediary role for body fat changes. This finding highlights a potential challenge for Asians, as fatty liver disease may progress without overt body fat changes, which are often used to screen for metabolic conditions among their White counterparts.

4.1. LIMITATIONS

This study has several limitations. First, the retrospective design inherently limits the ability to establish causal relationships between the variables studied. The study's modest sample size, particularly for Asian participants, may restrict statistical power and generalizability. Reliance on self-reported psychological and eating behavior measures can also introduce potential recall and social desirability biases. In addition, as the study was conducted in a single Southern California clinic, findings may not extend to other populations or settings with different demographic or healthcare characteristics. Finally, the use of a convenience sample in this cross-sectional analysis precludes the assessment of temporal relationships. Larger, longitudinal studies across diverse populations are needed to clarify the observed associations and their implications.

5. CONCLUSION

Disordered eating behaviors driven by impaired emotional functioning may act as a "silent killer" for Asians, contributing to the development of MASLD even in individuals with normal anthropometrics. Our findings highlight the need for tailored disease surveillance and prevention strategies, particularly among Asian populations, to address this risk.

For clinical practice, these results suggest that healthcare providers should consider screening for disordered eating behaviors and emotional health, even among patients who do not present with traditional metabolic risk factors. Early identification and targeted intervention focused on psychological well-being and eating patterns could play a crucial role in preventing MASLD in this population.

Future research should expand sample sizes to enhance the generalizability. Given that MASLD affects diverse groups differently, studies should aim to recruit robust, representative samples across ethnic and socioeconomic backgrounds. Longitudinal studies are needed to establish causal relationships and to understand the temporal dynamics between psychological factors, eating behaviors, and MASLD progression. By tracking patients over time, researchers can better discern the directionality and causative nature of these relationships. Longitudinal data would also allow for assessing how changes in psychological states or eating behaviors affect liver health over time, thereby identifying critical intervention points that reflect sociocultural factors (e.g., race, culture, age, and gender).

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CONFLICT OF INTEREST

The authors have no conflicts of interest to disclose.

AUTHOR CONTRIBUTIONS

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Writing–review & editing: Kelly A. Scheneman, Steven Cen, Tse-Ling Fong

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study is a retrospective chart review; the requirement for participant consent was waived by the Hoag Foundation IRB. This waiver was granted due to the minimal risk nature of the study and the use of de-identified data.

CONSENT FOR PUBLICATION

As this study involved only anonymized data with no identifying information, consent for publication was not required. All authors have reviewed and approved the final version of this manuscript for publication.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available upon reasonable request from the corresponding author. For any inquiries or to request the data, please contact: Kelly A. Scheneman (kelly.scheneman@hoag.org); Hoag Memorial Hospital Presbyterian, 3900 West Coast Hwy, Newport Beach, CA, US 92658.

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