

Original Article

Discrepancy between self-perceived dietary habits and objective clinical risk of liver fibrosis: A cross-sectional study of Japanese health checkup participants

Nao Kawabata PhD^{1,2}, Naoki Morimoto PhD³, Mizuho Shimada PhD⁴, Shizukiyo Ishikawa PhD⁵, Kentaro Kurashina PhD⁶

¹Department of Human Nutrition, Seitoku University, Chiba, Japan

²Department of Clinical Laboratory Medicine, Jichi Medical University, Tochigi, Japan

³Department of Medicine, Division of Gastroenterology, Jichi Medical University, Tochigi, Japan

⁴Jichi Medical University Health Care Center, Tochigi, Japan

⁵Center for Information, Jichi Medical University, Tochigi, Japan

⁶Department of Surgery, Division of Gastroenterological, General and Transplant Surgery, Jichi Medical University, Tochigi, Japan

Background and Objectives: Lifestyle modification is the cornerstone of liver disease management; however, its success depends on accurate self-assessment. We investigated the clinical characteristics of individuals at risk of liver fibrosis and the discrepancy between their objective risk and self-perceived lifestyle habits in patients with steatotic liver disease. **Methods and Study Design:** This cross-sectional study included 167 participants diagnosed with steatotic liver disease. Participants were categorized into a high-risk group ($n = 57$) and a control group ($n = 110$) based on the age-adjusted Fibrosis-4 index cutoffs. Multivariable logistic regression was used to identify independent factors, and self-perceived habits were compared to evaluate cognitive-clinical discrepancies. **Results:** Age was identified as the sole independent clinical factor associated with the risk of fibrosis. The high-risk group exhibited significantly lower alanine aminotransferase and triglyceride levels than those of the control group. Despite their higher clinical risk, the high-risk group reported significantly higher engagement in habitual exercise (53% vs. 31%, $p = 0.006$) and restorative sleep (odds ratio: 3.24). High-risk individuals were also significantly less likely to report the frequent intake of sweets (odds ratio: 0.253). **Conclusions:** A profound cognitive-clinical discrepancy exists among individuals at risk for liver fibrosis, which may lead them to misinterpret pathologically declining biomarkers as successful outcomes of their lifestyle efforts. Health promotion must move beyond general advice to incorporate perception-correcting feedback that reconciles subjective health views with objective pathological realities.

Key Words: liver fibrosis, Fibrosis-4 index, self-perception, dietary habits, weight management

INTRODUCTION

Liver fibrosis represents the critical histological determinant of long-term prognosis in chronic liver disease, serving as a precursor to cirrhosis, hepatocellular carcinoma, and increased cardiovascular mortality.¹ In the Asia-Pacific region, the etiology of liver disease has shifted from viral hepatitis to metabolic dysfunction-associated steatotic liver disease (MASLD), making early identification of liver fibrosis in the general population a public health priority.²⁻³ As liver biopsies are invasive, the Fibrosis-4 (FIB-4) index, calculated using age, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and platelet count, which was derived and validated in patients with nonalcoholic fatty liver disease or hepatitis C virus, is now a vital screening instrument in health checkup settings.⁴⁻⁷

Lifestyle modification, particularly weight management

through dietary intervention, is the primary treatment for preventing the progression of liver fibrosis.⁸ However, the success of these interventions depends on an individual's accurate self-perception of their behavior. While individuals with metabolic risk factors often exhibit a "perception gap" (e.g., underestimating energy intake),⁹⁻¹⁴ it remains unclear how those identified as "at-risk" via the FIB-4 index perceive their habits.

A critical challenge arises when individuals at high objective risk perceive their habits as already "healthy" or

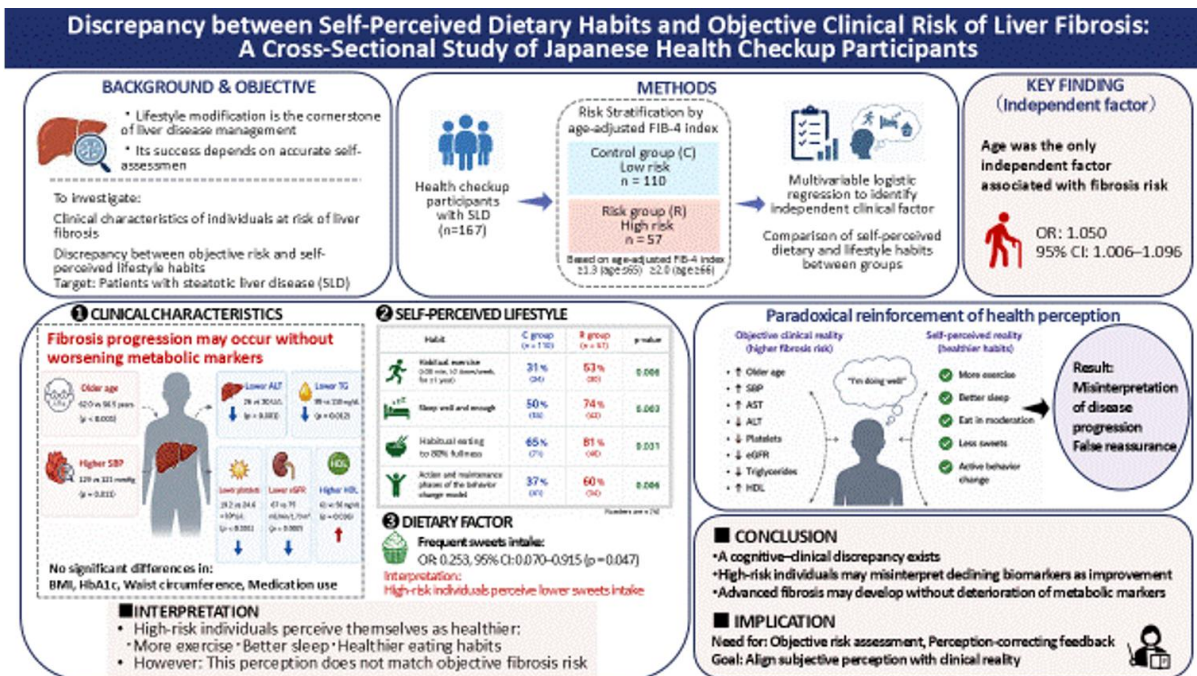
Corresponding Author: Dr. Nao Kawabata. Department of Human Nutrition, Seitoku University, 550 Iwase, Matsudo, Chiba 271-8555, Japan

Tel: +81-47-365-1111

Email: kawabata.nao@wa.seitoku.ac.jp; nkawabata@jichi.ac.jp

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Graphical abstract.

"sufficient." If high-risk patients believe they have already achieved successful lifestyle changes, they may ignore medical guidance.^{14–16} Previous clinical trends suggest that some biomarkers may paradoxically "improve" as liver fibrosis progresses,^{17,18} potentially reinforcing a false sense of security. Despite its importance, the extent of this "cognitive-clinical discrepancy" among those at risk of liver fibrosis has not been adequately investigated.

Therefore, this study aimed to elucidate the clinical characteristics of individuals with steatotic liver disease (SLD) at high risk for liver fibrosis and to investigate the discrepancy between their objective clinical risk and self-perceived lifestyle habits. By bridging this "cognitive-clinical discrepancy," we seek to provide a rationale for more effective perception-correcting nutritional interventions in the management of liver health.

METHODS

Study participants

This study included 2,157 individuals who underwent health checkups at the Jichi Medical University Health Screening Center between April and July 2023. Among them, 167 participants with SLD were included. The presence of SLD was determined through participants' documented medical history and a face-to-face medical interview conducted by trained physicians during the health checkup. Participants were operationally defined as having SLD if they reported a prior clinical diagnosis of SLD by a healthcare institution, were currently receiving medical treatment, or were under ongoing clinical follow-up observation for the condition. The participants were categorized into two groups based on the age-adjusted FIB-4 index cutoffs recommended by the MASLD Clinical Practice Guidelines^{19–21}: a high-risk group (R group, n = 57) defined as FIB-4 ≥ 1.3 for those ≤ 65 years of age and ≥ 2.0 for those ≥ 66 years of age, and a control group (C group, n = 110) (Figure 1).

Study design

This cross-sectional study used data from the Jichi Medical University Health Screening Center System, incorporating sex, age, body mass index (BMI), waist circumference, blood pressure, and comprehensive blood panels. Blood test parameters included fasting plasma glucose, hemoglobin A1c (HbA1c), total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, non-HDL cholesterol, triglycerides, uric acid, AST, ALT, γ -glutamyltransferase (γ -GTP), FIB-4 index, and estimated glomerular filtration rate (eGFR). The FIB-4 index was calculated as: $(\text{age} \times \text{AST}) / (\text{platelet count} [\times 10^4 / \mu\text{L}] \times \sqrt{\text{ALT}})$.

Study endpoints

There were two primary endpoints: first, to identify independent clinical determinants associated with an elevated risk of liver fibrosis in a health screening population, and second, to evaluate "cognitive-clinical discrepancy." This discrepancy was defined as the divergence between objective clinical risk (R and C groups) and participants' subjective self-perception of their dietary habits and physical activity levels.

Lifestyle and dietary assessment

Lifestyle habits were assessed using items extracted from the "Questionnaire Sheet for General Health Examinations" and a supplementary "Self-Perception of Dietary Intake" survey. The former is based on the standardized questionnaire recommended by the Japanese Ministry of Health, Labour and Welfare (MHLW) for national Specific Health Checkups ("Tokutei Kenshin"), which has been utilized in large-scale studies to identify metabolic risk factors.²² This questionnaire sheet assessed six domains: (1) smoking status, (2) energy balance, (3) physical activity/exercise level, (4) dietary behaviors related to metabolic syndrome risk, (5) quantity and quality of sleep, and (6) stages of the Transtheoretical Model of

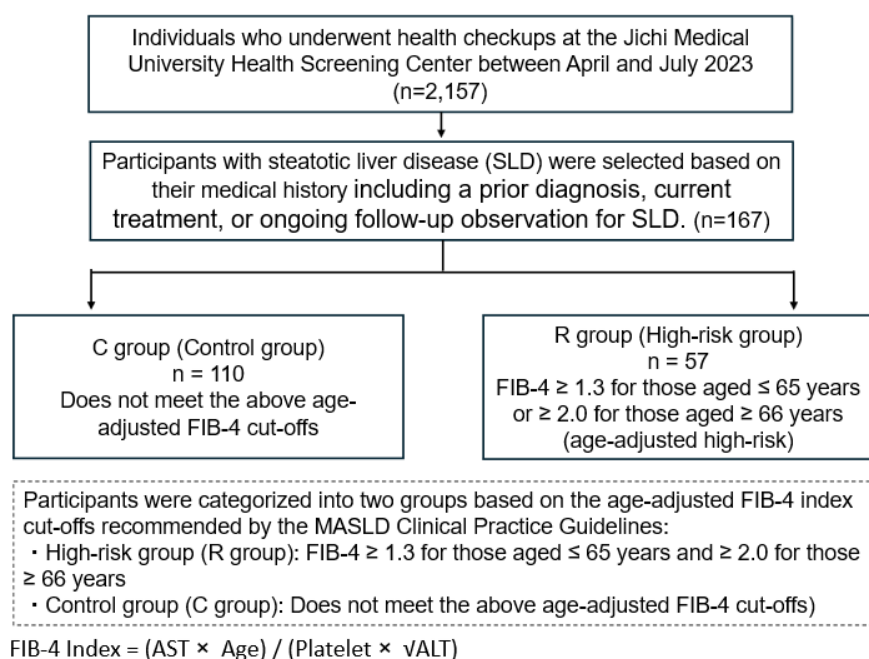


Figure 1. Flowchart of study participants. A total of 2,157 individuals underwent health checkups at the Jichi Medical University Health Screening Center between April and July 2023. A total of 167 participants with steatotic liver disease (SLD) were identified and included in the analysis. These participants were categorized into two groups based on the age-adjusted Fibrosis-4 (FIB-4) index cutoffs (≥ 1.3 for those ≤ 65 years of age and ≥ 2.0 for those ≥ 66 years of age): a high-risk group (R group, $n=57$) and a control group (C group, $n=110$).

Behavior Change. The stages of the Transtheoretical Model of Behavior Change were categorized as follows: a. pre-contemplation: no intention to improve; b. contemplation: intention to improve within 6 months; c. preparation: intention to improve within 1 month and have begun making small changes; d. action: actively working on improvement for less than 6 months; and e. maintenance: actively working on improvement for more than 6 months. Alcohol consumption was quantified based on responses to questions regarding "frequency of alcohol consumption (e.g., sake, shochu, beer, western liquor)" and "amount of alcohol consumed per day on drinking days." Participants reporting "every day" or "sometimes" for drinking frequency and "180–360 mL or more (based on 180 mL of sake)" for daily alcohol consumption were categorized as having an average daily pure alcohol intake of 40 g or more for males and 20 g or more for females.

The "Self-Perception of Dietary Intake" survey assessed intake frequency using a three-point scale (frequently, moderately, rarely) for the consumption of cereals, meat, seafood, eggs, soy products, vegetables, seaweed, fruits, milk/dairy products, fried and sautéed foods, salty foods, sweets, and sweet beverages. These 13 items were selected based on the standardized food group classifications used in the National Health and Nutrition Survey (NHNS) of Japan, which provides Content Validity for monitoring trends in the Japanese population. Specifically, these items represent major food groups (e.g., soy, seafood, and seaweed) and metabolic risk-related behaviors (e.g., intake of sweets and fried foods) defined by national standards. The methodological format of this specific survey as a simplified food group checklist has been validated in the Japanese population by Yatsuya et al.²³ and Tsuchihashi et al.,²⁴ demonstrating that such checklists provide reasonably good validity for ranking

individuals according to habitual intake. This tool was used pragmatically to capture the discrepancy between participant cognition and objective clinical status.

Statistical analysis

The sample size was determined based on data from previous domestic studies with parameters similar to those referenced in the literature.²⁵ It was assumed that the mean BMI of the hepatic fibrosis risk group was 24.5 ± 3.0 kg/m² and that of the control group was 23.0 ± 3.0 kg/m². A sample size of 64 participants per group was initially calculated as the target. However, since we included all eligible SLD cases identified during the study period, the final R group consisted of 57 participants, which was deemed sufficient for the primary comparisons as the total SLD population ($n = 167$) provided adequate representation.

Continuous variables are presented as mean $\pm$ standard deviation or median (interquartile range), and categorical variables as percentages. Normality was assessed using the Shapiro–Wilk test. Group comparisons between the R and C groups were conducted using the χ^2 test, t-test, or Mann–Whitney U test, as appropriate. To identify the independent determinants of liver fibrosis risk, a multivariate logistic regression analysis was performed using a stepwise method based on the likelihood ratio. Explanatory variables were selected based on their clinical relevance and univariate significance ($p < 0.05$). Model calibration and goodness-of-fit were evaluated using the Hosmer–Lemeshow test.

Since the FIB-4 index formula includes age, the associations between self-perceived food group intake and liver fibrosis risk were evaluated using age-adjusted logistic regression models to control for age-related confounding factors. Statistical significance was defined as a two-

tailed $p < 0.05$. All analyses were performed using SPSS Statistics version 21 (IBM Corp., Armonk, NY, USA).

Ethical considerations

This study was performed in accordance with the ethical standards of the 1964 Declaration of Helsinki and subsequent amendments. The study protocol was approved by the Jichi Medical University Clinical Research Ethics Committee (approval number: A23-086). The committee waived the requirement for informed consent due to the observational nature of this cross-sectional study utilizing pre-existing data. An opt-out mechanism was implemented via the Jichi Medical University Health Screening Center website, giving participants the opportunity to withdraw their consent.

RESULTS

Comparison of clinical background between control and risk groups

The clinical characteristics of participants in the control (C) group and the risk (R) group are summarized in Table 1.

Participants in group R were significantly older than those in group C (62.0 vs. 56.5 years, $p < 0.001$) and had a higher systolic blood pressure (SBP) (129 vs. 121 mmHg, $p = 0.011$). The R group exhibited significantly elevated AST levels (27 vs. 23 U/L, $p < 0.001$). Conversely, the R group had significantly lower ALT (26 vs. 30 U/L, p

< 0.001), platelet counts (19.2 vs. $24.6 \times 10^4/\mu\text{L}$, $p < 0.001$), and eGFR (67 vs. 75 mL/min/1.73 m², $p = 0.002$). In terms of lipid profiles, the R group showed significantly lower triglyceride levels (89 vs. 110 mg/dL, $p = 0.012$) and higher HDL cholesterol levels (61 vs. 56 mg/dL, $p = 0.036$). No significant differences were observed in BMI, waist circumference, γ -GTP, HbA1c, fasting plasma glucose, or the prevalence of medications between the groups.

Analysis of independent factors associated with liver fibrosis risk

To identify the determinants of liver fibrosis risk, we performed multivariable logistic regression analyses using three distinct models: clinical parameters, general lifestyle habits, and specific dietary intakes.

Clinical and biochemical model

In the model incorporating clinical and biochemical parameters (including blood pressure and laboratory data), age was identified as the sole independent factor significantly associated with the risk of liver fibrosis (odds ratio [OR]: 1.05, 95% confidence interval [CI]: 1.01–1.10) (Table 2). Other clinical markers, including BMI and lipid profiles, were not statistically significant in this model.

Table 1. Comparison of clinical background between control and risk groups

	Total (n=167)		p-value
	C group (n=110)	R group (n=57)	
Males, n (%)	84 (76)	39 (68)	0.269 [†]
Age (years)	56.5 (49.8, 65.3)	62.0 (58.0, 65.0)	<0.001
BMI (kg/m ²)	25.3 (23.0, 27.8)	24.5 (21.9, 28.1)	0.251
Waist circumference (cm)	90.9 (84.3, 97.4)	87.9 (80.4, 96.4)	0.178
SBP (mmHg)	121 (114, 133)	129 (120, 140)	0.011
DBP (mmHg)	77 (70, 83)	77 (74, 86)	0.324
AST (U/L)	23 (18, 29)	27 (23, 38)	<0.001
ALT (U/L)	30 (20, 43)	26 (19, 44)	<0.001
γ -GTP (U/L)	37 (25, 87)	34 (20, 65)	0.170
Platelets ($\times 10^4/\mu\text{L}$)	24.6 (22.1, 27.7)	19.2 (17.1, 22.7)	<0.001
HbA1c (%)	5.9 (5.6, 6.3)	6.0 (5.7, 6.5)	0.228
Fasting plasma glucose (mg/dL)	104 (97, 116)	108 (98, 126)	0.262
TC (mg/dL)	198 (178, 218)	198 (171, 216)	0.506
LDL cholesterol (mg/dL)	119 (103, 140)	116 (97, 134)	0.149
Non-HDL cholesterol (mg/dL)	141 (121, 158)	134 (106, 153)	0.069
HDL cholesterol (mg/dL)	56 (47, 64)	61 (49, 74)	0.036
Triglycerides (mg/dL)	110 (86, 156)	89 (70, 135)	0.012
Uric acid (mg/dL)	6.0 (5.0, 6.7)	5.7 (4.9, 6.6)	0.328
eGFR (mL/min/1.73 m ²)	75 (63, 82)	67 (57, 75)	0.002
Antihypertensive medications, n (%)	52 (47)	30 (53)	0.511 [†]
Antidiabetic medications, n (%)	17 (15)	15 (26)	0.091 [†]
Dyslipidemia medications, n (%)	39 (35)	24 (42)	0.400 [†]
FIB-4 index	0.98 (0.79, 1.24)	1.65 (1.45, 2.25)	--

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; AST, aspartate transaminase; ALT, alanine aminotransferase; γ -GTP, γ -glutamyltransferase; HbA1c, hemoglobin A1c; TC, total cholesterol; LDL cholesterol, low-density lipoprotein cholesterol; Non-HDL cholesterol, non-high-density lipoprotein cholesterol; HDL cholesterol, high-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate; FIB-4 index, fibrosis-4 index.

Data are presented for participants with steatotic liver disease (SLD) ($n = 167$). The risk group (R group) was defined based on age-adjusted FIB-4 index thresholds: ≥ 1.3 for individuals aged ≤ 65 years and ≥ 2.0 for those aged ≥ 66 years. The control group (C group) comprised participants with SLD whose values were below these age-adjusted thresholds.

Number of patients (%), median (interquartile range), Mann–Whitney test,

[†] χ^2 test of independence.

model, restorative sleep was identified as an independent

Table 2. Clinical factors associated with liver fibrosis risk

Variables	Odds ratio	95% confidence interval
Age	1.05	1.01, 1.10
SBP	1.02	0.992, 1.04
HDL cholesterol	1.01	0.990, 1.04
Triglycerides	0.994	0.987, 1.00
eGFR	0.972	0.945, 1.00

SBP, systolic blood pressure; HDL cholesterol, high-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate.

The multiple logistic regression model evaluated clinical factors associated with liver fibrosis risk in individuals with steatotic liver disease (SLD) (n = 167).

The model included the following explanatory variables: age, SBP, HDL cholesterol, triglycerides, and eGFR. These variables were selected based on significant differences or clinical relevance identified in univariate analysis.

Comparison of questionnaire sheet for general health examinations

Table 3 presents a comparison between the C and R groups in terms of lifestyle factors. To maintain a concise presentation, lifestyle variables derived from the Questionnaire Sheet for General Health Examinations were screened, and only those demonstrating statistically significant differences between the two groups were compiled in Table 3. Relative to the C group, the R group had a significantly higher proportion of individuals who engaged in habitual exercise (53% vs. 31%, $p = 0.006$) and stopped eating before 80% of fullness (ate in moderation) (81% vs. 65%, $p = 0.031$). Furthermore, the R group had a significantly higher rate of restorative sleep (74% vs. 50%, $p = 0.003$). Regarding the stages of the Transtheoretical Model of Behavior Change, the R group had a significantly higher percentage of participants in the action or maintenance stages (60% vs. 37%, $p = 0.006$). No significant differences were found between the groups in terms of smoking status or energy balance.

Multivariable analysis of lifestyle factors

A separate multivariable logistic regression analysis was performed to identify independent lifestyle determinants associated with the risk of liver fibrosis (Table 4). In this

factor significantly associated with the high-risk group (OR: 3.24, 95% CI: 1.50–7.03), as well as age. Other factors, including male sex, BMI, moderate eating, and habitual exercise, did not show statistically significant associations in this multivariable model.

Self-perceived dietary intake model

Table 5 shows the association between self-perceived food intake and the risk of liver fibrosis, adjusted for sex, age, and BMI. Multivariable analysis revealed that participants in the high-risk group were significantly less likely to report a frequent intake of sweets (OR: 0.253, 95% CI: 0.070–0.915, p for trend = 0.047). No other food items, including cereals, meat, seafood, vegetables, or beverages, were significantly associated with the risk of liver fibrosis in the adjusted model.

DISCUSSION

This study aimed to ascertain the clinical characteristics of individuals at risk for liver fibrosis based on the FIB-4 index among patients with SLD and examine the discrepancy between their objective clinical risk and self-perceived lifestyle habits. In our multivariable analysis, age was identified as the sole independent clinical determinant associated with an elevated risk of fibrosis.

Table 3. Comparison of lifestyle factors between the control and risk groups

Category	Questions	Total (n=167)		
		C group (n=110)	R group (n=57)	p-value
Physical activity/exercise level	Are you in a habit of doing exercise to sweat lightly for over 30 minutes a time, 2 times weekly, for over a year? Numbers indicate number of "yes" responses (%)	34 (31)	30 (53)	0.006
Dietary behaviors related to metabolic syndrome risk	Do you stop eating before 80% of fullness (eat in moderation)? Numbers indicate number of "yes" responses (%)	71 (65)	46 (81)	0.031
Quantity and quality of sleep	Do you sleep well and enough? Numbers indicate number of "yes" responses (%)	55 (50)	42 (74)	0.003
Stages of the Transtheoretical Model of Behavior Change	Action and maintenance phases of the behavior change model Numbers indicate number of "yes" responses (%)	41 (37)	34 (60)	0.006

Data are presented for participants with steatotic liver disease (SLD) (n=167). The risk group (R group) was defined based on age-adjusted FIB-4 index thresholds: ≥ 1.3 for individuals aged ≤ 65 years and ≥ 2.0 for those aged ≥ 66 years. Number of persons (%), χ^2 test of independence. Only items with statistically significant differences are displayed to keep the table concise.

No significant differences were observed between groups for the following lifestyle factors: current smoking status, weight gain of over 10 kg since age 20, daily physical activity or walking for more than one hour, walking speed, eating speed, late-night supper (within two hours before bedtime), snacking or drinking sweet beverages between meals, skipping breakfast, and alcohol consumption (exceeding 40 g/day for males and 20 g/day for females).

Table 4. Multivariable analysis of self-perceived lifestyle habits and liver fibrosis risk

Variables	Odds ratio	95% confidence interval
Male	0.514	0.225, 1.17
Age	1.07	1.03, 1.12
BMI	1.02	0.942, 1.11
Habitual eating to 80% fullness	1.95	0.852, 4.45
Habitual exercise	1.59	0.751, 3.36
Restorative sleep	3.24	1.50, 7.03

BMI, body mass index. Multivariable logistic regression models were used to identify self-perceived lifestyle factors independently associated with liver fibrosis risk in individuals with steatotic liver disease (SLD) (n = 167).

Explanatory variables included male sex, age, BMI, habitual eating to 80% fullness, habitual exercise (≥ 30 minutes per session), and restorative sleep. These variables were selected based on their statistical significance or clinical relevance established in univariate analysis.

Table 5. Multivariable analysis of self-perceived dietary intake and liver fibrosis risk

Food item	Rarely OR (Reference)	Moderately OR (95% CI)	Frequently OR (95% CI)	p for trend
Cereals	1.00	3.17 (0.626, 16.0)	2.70 (0.500, 14.6)	0.599
Meat	1.00	0.369 (0.083, 1.65)	0.264 (0.052, 1.34)	0.143
Seafood	1.00	1.24 (0.455, 3.36)	1.85 (0.474, 7.26)	0.386
Eggs	1.00	1.32 (0.347, 5.02)	1.93 (0.493, 7.52)	0.233
Soy products	1.00	2.21 (0.582, 8.41)	2.68 (0.663, 10.8)	0.208
Vegetables	1.00	1.41 (0.264, 7.53)	1.83 (0.341, 9.85)	0.356
Seaweed	1.00	1.93 (0.885, 4.20)	2.47 (0.690, 8.86)	0.080
Fruits	1.00	0.582 (0.268, 1.26)	0.558 (0.187, 1.67)	0.706
Milk/Dairy products	1.00	1.31 (0.464, 3.69)	1.22 (0.415, 3.58)	0.823
Fried and sautéed foods	1.00	1.34 (0.351, 5.13)	0.636 (0.142, 2.84)	0.213
Salty foods	1.00	0.746 (0.358, 1.55)	0.619 (0.139, 2.75)	0.386
Sweets	1.00	0.884 (0.371, 2.10)	0.253 (0.070, 0.915)	0.047
Sweet beverages	1.00	0.845 (0.392, 1.82)	0.720 (0.207, 2.51)	0.538

SLD, steatotic liver disease; BMI, body mass index; OR, odds ratio; CI, confidence interval.

Multivariable logistic regression analysis was performed to evaluate the association between the self-perceived frequency of intake for each food item and the risk of liver fibrosis among participants with SLD (n = 167).

The high-risk group was defined by age-adjusted FIB-4 index thresholds (≥ 1.3 for individuals aged ≤ 65 years and ≥ 2.0 for those aged ≥ 66 years).

For each food item, the 'rarely' category was designated as the reference group (OR = 1.00), and adjusted ORs (95% CIs) were calculated for 'moderately' and 'frequently' categories, with adjustments for sex (male), age, and BMI.

p for trend was determined by treating the three-point intake frequency as an ordinal variable (scores: rarely = 0, moderately = 1, frequently = 2) within the same multivariable-adjusted model.

Notably, while the high-risk (R) group was significantly older and exhibited higher SBP, other metabolic parameters such as BMI, HbA1c, and the prevalence of medication use did not differ significantly between the R and C groups. This suggests that in individuals already diagnosed with SLD, the progression of liver fibrosis may not always be accompanied by an obvious escalation of current metabolic markers, making the identification of high-risk patients through standard checkups more challenging.

In the present study, the lower ALT levels in the R group likely reflect a mathematical selection bias inherent to the FIB-4 index, where ALT is positioned in the denominator. In the original derivation of the FIB-4 index, the odds ratio for ALT was reported as 0.493, indicating that higher ALT levels were inversely associated with advanced fibrosis.⁴ This structural design captures the known pathological transition from an ALT-dominant pattern in early stage steatosis to an AST-dominant pattern (AST/ALT ratio > 1) as liver fibrosis progresses.²⁶ In our cohort, the AST/ALT ratio reversed from 0.77 in the C group to 1.04 in the R group, supporting this pathological shift. Therefore, the lower ALT values in the R group should not be interpreted as a genuine metabolic 'im-

provement' but rather as a marker of advanced disease captured by the inverse relationship between ALT and fibrosis in the FIB-4 formula.

The most significant finding of this study is the profound "cognitive-clinical discrepancy," where individuals at high risk for liver fibrosis perceived their lifestyle as healthier than those at lower risk. Individuals in the R group, who are genuinely engaged in the "action" stage of behavior change, may find their efforts falsely validated by these declining biomarkers, thereby masking the objective reality of their liver fibrosis risk. Our findings demonstrating that the R group maintained anthropometric profiles similar to those of the C group while reporting significantly higher levels of physical activity suggest that the discrepancy is not between "effort and energy balance," but between "subjective effort and the biological reality of liver fibrosis." The sense of accomplishment from lifestyle changes, reinforced by misleadingly "normal" blood markers, prevents individuals from recognizing the persistent urgency of their liver conditions.

These findings imply that standard dietary advice may be ineffective if high-risk patients believe that they are already following it or have already achieved "success"

through their efforts. Effective health promotion requires perception-correcting counseling. Dietitians and primary care providers should move beyond general instructions and utilize objective clinical surrogates, such as the age-adjusted FIB-4 index and the paradoxical decline of ALT and triglycerides, to highlight the gap between a patient's perceived efforts and their biological reality. Rather than simply praising "normal" laboratory values, clinicians must explain how these trends can be deceptive in the context of advancing liver fibrosis, thereby helping patients recognize the ongoing need for specialized medical care despite their subjective clinical "improvement."

This study had several limitations. First, lifestyle data were based on self-reported questionnaires, which are subject to memory errors and social desirability bias.²⁷ In addition, these self-reports may also be influenced by a confirmation bias. Participants in the R group may have over-reported healthy behaviors because they genuinely believed that their "improved" lipid and ALT levels reflected successful lifestyle modifications. Future studies should incorporate objective measurements, such as dietary photography and accelerometers, to more accurately quantify the discrepancy between perception and reality.

Second, the risk of liver fibrosis was evaluated using the FIB-4 index rather than gold-standard methods such as liver biopsy or elastography. Although age-adjusted cutoffs were applied to improve diagnostic accuracy, the FIB-4 index remains a surrogate marker and may not fully reflect the histological state of the liver, particularly in a general screening population in which advanced liver fibrosis is relatively uncommon. Furthermore, the small overall sample size of participants with SLD ($n=167$) and the subsequent subdivision into the R group ($n=57$) and C group ($n=110$) introduced critical constraints on our statistical power. Specifically, in the multivariable logistic regression analyses, adjusting for multiple covariates (such as sex, age, and BMI) relative to the limited number of participants in each group likely rendered the statistical models underpowered. This methodological restriction increases the susceptibility to Type II errors, meaning that the study may have lacked sufficient power to detect true baseline differences or subtle associations regarding certain dietary habits. Consequently, the non-significant findings observed for several food items must be interpreted with necessary caution due to this limited statistical power.

Third, the cross-sectional design of this study precludes the establishment of a causal relationship. It remains unclear whether the lower lipid levels observed in the R group were the result of intentional dietary efforts or a paradoxical consequence of advancing liver fibrosis. Furthermore, we cannot rule out the possibility that participants in the high-risk group had already initiated healthy lifestyle and dietary modifications after becoming aware of their diagnosis or clinical condition. Such post-diagnosis behavioral changes provide a highly plausible alternative explanation for the observed discrepancies between self-perceived habits and objective clinical risk; that is, the favorable dietary perceptions in the high-risk group may reflect recent behavioral improvements rather than a true baseline cognitive mismatch. This potential for reverse causality, where the clinical condition or prior

clinical awareness influences the perception and reporting of lifestyle behaviors, represents an important challenge that should be addressed in future longitudinal studies.

Fourth, the presence of SLD was defined based on participants' documented medical history and physician interviews rather than mandatory objective modalities, such as abdominal ultrasonography, computed tomography, or liver biopsy, for all participants. Although this interview-based approach is pragmatic and widely accepted in large-scale general health screening settings, it might have introduced a classification bias or led to an underestimation of asymptomatic, undiagnosed SLD cases in the control population. Consequently, our findings should be interpreted within the context of a real-world epidemiological screening rather than strict radiological or histological staging.

Finally, although the R group demonstrated higher levels of action in behavior change, it remains uncertain whether individualized interventions by dietitians can reduce the risk of liver fibrosis by correcting patients' misinterpretations of clinical data. Future research should examine whether perception-correcting counseling strategies can improve long-term outcomes and contribute to a true reduction in the risk of liver fibrosis.

Conclusions

In conclusion, the risk of liver fibrosis is not always accompanied by an obvious deterioration in metabolic markers; rather, it may be masked by paradoxical improvements in ALT and lipid profiles associated with disease progression and the mathematical characteristics of non-invasive screening tools. These misleading clinical trends can lead high-risk individuals to misinterpret their pathological conditions as the result of successful lifestyle behaviors. Therefore, early intervention by healthcare providers, particularly dietitians, is essential. Beyond promoting healthy habits, counseling should focus on correcting misperceptions of clinical data to prevent patients from overlooking the progression of liver fibrosis until it reaches an advanced and potentially irreversible stage.

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CONFLICT OF INTEREST AND FUNDING DISCLOSURES

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