Non-alcoholic fatty liver disease (NAFLD) is the most common chronic liver disease, and its pathogenesis is complicated and triggered by unbalanced diet, sedentary lifestyle, and genetic background. The aim of this study was to construct and validate a nomogram incorporated lifestyle habits for predicting NAFLD incidence.

The overall cohort was divided into training set and test set as using computer-generated random numbers. We constructed the nomogram by multivariate logistic regression analysis in the training set. Thereafter, we validated this model by concordance index, the area under the receiver operating characteristic curve (ROC), net reclassification index, and a calibration curve in the test set. Additionally, we also evaluated the clinical usefulness of the nomogram by decision curve analysis.

There were no statistically significant differences about characteristics between training cohort (n = 748) and test cohort (n = 320). Eleven features (age, sex, body mass index, drinking tea, physical exercise, energy, monounsaturated fatty acids, polyunsaturated fatty acids, hypertension, hyperlipidemia, diabetes) were incorporated to construct the nomogram, concordance index, the area under the ROC curve, net reclassification index were 0.801, 0.801, and 0.084, respectively, indicating the nomogram have good discrimination of predicting NAFLD incidence. Also, the calibration curve showed good consistency between nomogram prediction and actual probability. Moreover, the decision curve showed that when the threshold probability of an individual is within a range from approximately 0.5 to 0.8, this model provided more net benefit to predict NAFLD incidence risk than the current strategies.

This nomogram can be regarded as a user-friendly tool for assessing the risk of NAFLD incidence, and thus help to facilitate management of NAFLD including lifestyle and medical interventions.

**Abbreviations:** AUC = area under receiver operating characteristic curve, BMI = body mass index, C-index = Concordance index, DCA = decision curve analysis, MetS = metabolic syndrome, MUFAs = monounsaturated fatty acids, NAFLD = non-alcoholic fatty liver disease, NRI = net reclassification index, PUFAs = polyunsaturated fatty acids, ROC = receiver operating characteristic curve.

**Keywords:** nomogram, nonalcoholic fatty liver disease, predictive modeling, risk factor
Although several models have been developed to predict NAFLD risk,\cite{11,12} a simple nomogram incorporated lifestyle indicators has not been developed to assess NAFLD incidence. In the present study, we sought to develop and validate a nomogram for predicting NAFLD risk, and provide an individualized prediction tool by cost-benefit variables. The predictive value of the model was evaluated based on discrimination, calibration, and clinical utility in the training and validation sets. This simple-to-use model might serve as an early warning and prediction system for us.

### 2. Methods and materials

#### 2.1. Participants

The raw data was downloaded from the Dryad Digital Repository (http://www.datadryad.org/) for secondary analysis, which were shared by Chen et al.\cite{10} This retrospective cohort study included 534 NAFLD cases and 534 controls. All participants were randomly split into 2 groups in a 7:3 ratio. Thus, 748 participants were allocated to the training group, and 320 participants were allocated to the test group. NAFLD was newly diagnosed by liver ultrasonography, excluding alcoholic hepatitis, autoimmune hepatitis, viral hepatitis, or drug-induced liver disease. Participants were excluded as follows: age <18 or >70 years; taking lipiodlowering or weight loss drugs; did not answer food frequency and nut intake questionnaires. The controls were randomly selected and were frequency-matched by age, sex, ethnicity, and region. The raw data contained age, sex, body mass index (BMI), education level, marital status, income, occupation, smoking, drinking tea, physical exercise, history of hyperlipidemia, diabetes, and hypertension. Daily nut and energy intake were calculated from a semiquantitative food frequency questionnaire. Monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs) were calculated by multiplying the nutrient content of the specified portion by frequency and summing across all relevant food items. Ethical approval was not necessary as the data available in a public website.

#### 2.2. Construction of the nomogram

Bivariate univariate logistic regression analysis was used to select the features at $P<.05$. Next, identified features were then included in a multivariate logistic regression analysis using a stepwise forward selection. Finally, we developed a nomogram to estimate NAFLD incidence in the training group.

#### 2.3. Discrimination of the nomogram

Concordance index (C-index) and the area under receiver operating characteristic curve (AUC) were applied to assess the discrimination of the nomogram in the test cohort, and their value $>0.7$ suggested good discrimination. The net reclassification index (NRI) was computed to evaluate the predictive capabilities of the 2 models. Additionally, a calibration plotting was used to evaluate the agreement between actual probability and nomogram prediction.

#### 2.4. Decision curve analysis (DCA) of the nomogram

We performed DCA to evaluate the clinical application of the model in the test cohort. DCA is an evaluation method that calculates the net benefits of predictive models.\cite{13}

#### 2.5. Identification of cutoff value for continuous variables

We performed receiver operating characteristic curve (ROC) analysis to evaluate the performance and optimal cutoff values of identified variables for NAFLD incidence in the whole cohort. The performance of variables for predicting in NAFLD was defined by AUC. The optimal cutoff values were defined by the highest Youden index (sensitivity + specificity $-1$).

#### 2.6. Statistical analyses

Measurement data were expressed as the means±standard deviation and analyzed by $t$ tests between the 2 groups. Numerical data were expressed as percentages and analyzed by chi-square test. Logistic regression analysis, nomogram, C-index, ROC, NRI, calibration curve, and DCA curve were conducted in R “stats,” “pROC,” “rms,” “nrcens,” “Resourcselection,” and “rmda” packages. All statistical tests were two-sided with P-values $<.05$ considered to indicate a statistically significant difference.

### 3. Results

#### 3.1. Participant characteristics

There are 728 men (68.2%) and 340 women (31.8%) in the overall cohort, and most (63.1%) were aged 40 to 60 years. Among all participants, nut daily intake was 8.01±12.97 g/d, energy was 2215.79±609.74 kcal/d, MUFA daily intake was 32.64±8.97 g/d, and PUFA daily intake was 24.62±4.90 g/d. The overall cohort (n=1068) was divided into training cohort (n=748) and test cohort (n=320) according to the ratio of 7:3. The participant characteristics including demographics, lifestyle habits, history of diseases, and nut intake are summarized in Table 1. Significant differences of characteristics were not observed between these 2 cohorts.

#### 3.2. Risk factor selection

Univariate logistic regression analysis identified 7 candidate features including BMI, drinking tea, physical exercise, hypertension, energy, MUFAs, and PUFAs intake (Fig. 1). To adjust various confounding factors, we performed multivariate logistic regression analysis to further explore risk factors predisposed to NAFLD. The results revealed that BMI and PUFAs daily intake are independent risk factors of NAFLD. Individuals with BMI $\geq 24.0$ were also 6.391 times more likely to develop NAFLD than those BMI $<24.0$. Each increase in PUFAs daily intake by 1g increased the risk of developing NAFLD by 1.141% (Table 2).

#### 3.3. Construction of the nomogram

The nomogram was developed to analyze NAFLD incidence incorporated the significant features (BMI, drinking tea, physical exercise, hypertension, energy, MUFAs, and PUFAs intake) and well-known factors (age, sex, hyperlipidemia, diabetes) in the training group (Fig. 2).

#### 3.4. Evaluation of the nomogram

We next evaluated the predictive model using various metrics in the test cohort. A C-index was 0.801, 95% CI, 0.738 to 0.864 which demonstrated moderate accuracy of the nomogram. The
AUC was 0.801, and the cutoff was 0.189 (Fig. 3), indicating a good discrimination of the nomogram. The calibration curve showed excellent performance of the predictive model (Fig. 4). To further explore whether independent risk (PUFAs intake) exerts an additional effect on this model, we compared model B (age, sex, BMI, drinking tea, physical exercise, energy, MUFAs intake, PUFAs intake, history of hyperlipidemia, diabetes, and hypertension) with model A (age, sex, BMI, drinking tea, physical

### Table 1
Baseline characteristics of training, test, and overall cohorts.

| Characteristics       | Training cohort (n = 748) | Test cohort (n = 320) | Overall cohort (n = 1068) | P value |
|-----------------------|--------------------------|-----------------------|---------------------------|---------|
| Sex                   |                          |                       |                           |         |
| Male                  | 504 (67.4%)              | 224 (70.0%)           | 728 (68.2%)               | .441    |
| Female                | 244 (32.6%)              | 96 (30.0%)            | 340 (31.8%)               |         |
| Age, y                |                          |                       |                           |         |
| <40                   | 188 (25.1%)              | 90 (28.1%)            | 278 (26.0%)               | .494    |
| 40–60                 | 475 (63.5%)              | 199 (62.2%)           | 674 (63.1%)               |         |
| ≥60                   | 85 (11.4%)               | 31 (9.7%)             | 116 (10.9%)               |         |
| Education             |                          |                       |                           | .394    |
| Primary school and less than | 63 (8.4%)         | 26 (8.1%)            | 89 (8.3%)                 |         |
| Junior middle and high school | 307 (41.0%)     | 118 (36.9%)          | 425 (39.8%)               |         |
| Junior college or above | 378 (50.5%)    | 176 (55.0%)          | 554 (51.9%)               |         |
| Marriage              |                          |                       |                           | .411    |
| Single                | 82 (11.0%)               | 29 (9.1%)             | 111 (10.4%)               |         |
| Married or other      | 666 (89.0%)              | 291 (90.9%)           | 957 (89.6%)               |         |
| BMI, kg/m²             |                          |                       |                           | .842    |
| <24.0                 | 411 (54.9%)              | 173 (54.1%)           | 584 (54.7%)               |         |
| ≥24.0                 | 337 (45.1%)              | 147 (45.9%)           | 484 (45.3%)               |         |
| Income (yuan/mo)      |                          |                       |                           | .764    |
| <2000                 | 45 (6.0%)                | 22 (6.9%)             | 67 (6.3%)                 |         |
| 2000–3000             | 229 (30.6%)              | 102 (31.9%)           | 331 (31.0%)               |         |
| ≥3000                 | 474 (63.4%)              | 196 (61.3%)           | 670 (62.7%)               |         |
| Smoke                 |                          |                       |                           | .538    |
| No                    | 530 (70.9%)              | 220 (68.8%)           | 750 (70.2%)               |         |
| Yes                   | 218 (29.1%)              | 100 (31.3%)           | 318 (29.8%)               |         |
| Drinking tea          |                          |                       |                           | .564    |
| No                    | 308 (41.2%)              | 125 (39.1%)           | 433 (40.5%)               |         |
| Yes                   | 440 (58.8%)              | 195 (60.9%)           | 635 (59.5%)               |         |
| Occupation            |                          |                       |                           | .831    |
| Mental labor          | 213 (28.5%)              | 97 (30.3%)            | 310 (29.0%)               |         |
| Physical labor        | 174 (23.3%)              | 73 (22.8%)            | 247 (23.1%)               |         |
| Other                 | 361 (48.3%)              | 150 (46.9%)           | 511 (47.8%)               |         |
| Physical exercise     |                          |                       |                           | .279    |
| Light                 | 235 (31.4%)              | 115 (35.9%)           | 350 (32.8%)               |         |
| Moderate              | 218 (29.1%)              | 93 (29.1%)            | 311 (29.1%)               |         |
| Severe                | 296 (39.4%)              | 112 (35.0%)           | 407 (38.1%)               |         |
| Hyperlipidemia        |                          |                       |                           | .954    |
| No                    | 713 (95.3%)              | 306 (95.6%)           | 1019 (95.4%)              |         |
| Yes                   | 35 (4.7%)                | 14 (4.4%)             | 49 (4.6%)                 |         |
| Diabetes              |                          |                       |                           | .606    |
| No                    | 724 (96.8%)              | 307 (95.0%)           | 1031 (96.5%)              |         |
| Yes                   | 24 (3.2%)                | 13 (4.1%)             | 37 (3.5%)                 |         |
| Hypertension          |                          |                       |                           | .184    |
| No                    | 577 (77.1%)              | 234 (73.1%)           | 811 (75.9%)               |         |
| Yes                   | 171 (22.9%)              | 86 (26.9%)            | 257 (24.1%)               |         |
| Nut frequency         |                          |                       |                           | .735    |
| None                  | 77 (10.3%)               | 37 (11.6%)            | 114 (10.7%)               |         |
| 1–3 times/d           | 45 (6.0%)                | 24 (7.5%)             | 69 (6.5%)                 |         |
| 1–2 times/wk          | 208 (27.8%)              | 95 (29.7%)            | 303 (28.4%)               |         |
| 3–6 times/wk          | 55 (7.4%)                | 25 (7.8%)             | 80 (7.5%)                 |         |
| 1–3 times/mo          | 206 (27.5%)              | 81 (25.3%)            | 287 (26.9%)               |         |
| <3 times/mo           | 157 (21.0%)              | 58 (18.1%)            | 215 (20.1%)               |         |
| Nut daily intake, g/d |                          |                       |                           | .204    |
| Energy, kcal/d        | 2215.92±606.29           | 2215.48±618.67        | 2215.79±609.74            | .991    |
| MUFA daily intake, g/d| 32.66±9.07               | 32.61±8.73            | 32.64±8.97                | .939    |
| PUFAs daily intake, g/d| 24.51±4.88             | 24.86±4.94            | 24.62±4.90                | .290    |

Data are shown as numbers (%) or mean±SD. BMI= body mass index, MUFA= monounsaturated fatty acids, PUFAs= polyunsaturated fatty acids.


exercise, energy, MUFAs intake, history of hyperlipidemia, diabetes, and hypertension). The probability 0.189 from ROC analysis was used as thresholds for categorical NRI. The value of NRI was 0.084, 95% CI, −0.005 to 0.089 indicating that predicted risks have not been reclassified in the old and new models. Collectively, these results revealed that the nomogram shows good performance of predicting NAFLD incidence.

### 3.5. Clinical usefulness of nomogram

The DCA of the nomogram is presented in Fig. 5. The decision curve showed that when the threshold probability of an individual is within a range from approximately 0.5 to 0.8, this model provided more net benefit to predict NAFLD incidence risk than the “all” or “none” strategies.
3.6. Cutoff value for continuous variables

The AUC for energy, MUFAs intake, and PUFAs intake was 54.28, 60.02, 67.66, respectively, indicating that PUFAs intake contribute the most to NAFLD incidence (Fig. 6). The cutoff values of energy, MUFAs intake, and PUFAs intake were 2142.08kcal/d, 31.42, and 24.66g/d, respectively, to optimally predict the risk of NAFLD (Table 3).

4. Discussion

In this retrospective cohort study, we constructed a novel tool for predicting NAFLD incidence using easily available variables including demographic characteristics (age, sex), lifestyle habits (drinking tea, physical exercise, energy, MUFAs, and PUFAs intake), and physical examination parameters (BMI, history of hypertension, hyperlipidemia, diabetes, and hypertension). We also validated this model using C-index, AUC, NRI, and calibration curve, indicating good performance of predicting NAFLD incidence. Moreover, the nomogram was demonstrated to have good clinical usefulness for predicting NAFLD incidence.

BMI is the most commonly used to define an individual as underweight, normal weight, overweight, or obesity. BMI $\geq$ 24 kg/m$^2$ is considered overweight, BMI $\geq$ 28 kg/m$^2$ is considered obesity for Chinese.[14] Obesity is not only a well-known risk factor for the development of NAFLD, but also linked with progression of liver disease.[15] A retrospective longitudinal cohort study revealed that BMI was positively correlation with NAFLD incidence, and it was considered as the most useful predictive risk factor for NAFLD in both sexes.[16] Another study indicated that increase in BMI during young adulthood correlates with a greater risk of NAFLD in midlife.[17] Consistent with previous research, our results also implied BMI is an independent risk factor for NAFLD. BMI incorporated our NAFLD-predictive model was reliable and accurate parameter.

Our study revealed that hypertension is a risk factor for NAFLD onset. Evidence demonstrated that individuals with hypertension have a higher prevalence of NAFLD, and the risk of NAFLD is independently associated with hypertension and blood pressure category.[18] Similarly, Donati et al[19] reported that hypertensive patients had a significantly higher prevalence of NAFLD, which may be related to increases in insulin resistance and BMI. MetS is a cluster of metabolic abnormalities, comprising obesity, hypertension, dyslipidemia, hyperglycemia, and insulin resistance. A possible explanation is that hypertension, a key factor for MetS, might indirectly NAFLD onset; however, their causal association would still have to be clearly determined.

It is well established that excess caloric intake lead to obesity and insulin resistance, and thus a leading risk factor for

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| Traits                  | OR (95%CI)  | P value |
|-------------------------|-------------|---------|
| BMI $\geq$24.0          | 6.391 [4.552–8.972] | .000    |
| Tea                     | 1.026 [0.727–1.447]  | .885    |
| [0,1-3]Moderate exercise| 0.770 [0.502–1.182] | .232    |
| Severe                  | 0.842 [0.562–1.261]  | .403    |
| Hypertension            | 1.281 [0.862–1.903]  | .220    |
| Energy                  | 1.000 [0.999–1.000]  | .066    |
| MUFAs daily intake      | 1.007 [0.983–1.031]  | .581    |
| PUFAs daily intake      | 1.141 [1.092–1.192]  | .000    |

BMI = body mass index, MUFAs = monounsaturated fatty acids, PUFAs = polyunsaturated fatty acids.
NAFLD. Clearly, gradual weight loss achieved by energy restriction regardless of physical activity condition, could improve liver fat deposit, insulin resistance, and hepatic inflammation and fibrosis. Recent studies have also noted that lifestyle change including energy restriction and increased physical activity during 6 to 12 months reduces liver fat and volume, steatosis, and incident NAFLD. These results corroborate the findings of our work in association between lifestyle and NAFLD. One surprising result was that drinking tea was found to be inversely associated with NAFLD incidence. A meta-analysis of the clinical trials found that green tea has a favorable effect on BMI, liver enzymes, and blood lipids parameters. This may be the mechanism by reducing dietary lipid absorption, lipogenesis, hepatic gluconeogenesis, and lipid peroxidation levels.

Nuts predominantly contain unsaturated fatty acids (MUFAs and PUFAs) and have a relatively low amount of saturated fatty acids. Frequent nut consumption could have favorable effect on lipid metabolism and endothelial function, and thus reduce risk of cardiovascular disease. A case-control study found that low intake of nuts was associated with a significantly higher risk for NAFLD in Korean men. Nuts, component of mediterranean diet pattern, are dietary recommendations for the prevention and management of NAFLD in adults. The high energy density and fat content of nuts has raised concerns that regular nut intake will lead to weight gain. For example, Alper and Mattes reported that peanut consumption for 8 weeks contributed to body weight gain (1 kg). In the present study, we estimated the optimal cutoff values of energy and unsaturated fatty acids intake to predict NAFLD incidence, which may provide references in defining the best thresholds of nut intake for the Chinese individuals.

There are several limitations to need to be acknowledged in the present study. First, this study is a case-control study design, selection bias and recall bias are inherent weaknesses. The prospective, longitudinal study design is needed to confirm these findings. Second, detailed food intake questionnaires were not available, such as different types of nuts and tea. Third, although the performance of our nomogram was evaluated with internal validation in the same population, there is a lack of external verification in other regions and countries.

Taken together, we constructed a nomogram incorporated 9 risk predictors (age, sex, drinking tea, physical exercise, energy, MUFAs, PUFAs intake, BMI, history of hypertension, hyperlip-
Table 3: Optimal cutoff values of identified risk factors for NAFLD.

| Variable          | Cutoff value | Sensitivity (%) | Specificity (%) | Youden Index |
|-------------------|--------------|----------------|-----------------|--------------|
| Energy, kcal/d    | 2142.1       | 56.4           | 52.2            | 0.076        |
| MUFAs, g/d        | 31.4         | 57.7           | 60.5            | 0.182        |
| PUFAs, g/d        | 24.7         | 62.4           | 68.0            | 0.304        |

MUFAs = monounsaturated fatty acids, NAFLD = nonalcoholic fatty liver disease, PUFA = polyunsaturated fatty acids.

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Author contributions

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References

[1] Zelber-Sagi S, Ivanovsky-Wajcman D, Fliss Isakov N, et al. High red and processed meat consumption is associated with non-alcoholic fatty liver disease and insulin resistance. J Hepatol 2018;68:1239–46.

[2] Younossi ZM, Koenig AB, Abdellatif D, Fazely Z, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease-meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology 2016;64:73–84.

[3] Adams LA, Anstee QM, Tilg H, Targher G. Non-alcoholic fatty liver disease and its relationship with cardiovascular disease and other extra-hepatic diseases. Gut 2017;66:1138–53.

[4] Stefan N, Haring HU, Cusi K. Non-alcoholic fatty liver disease: causes, diagnosis, cardiometabolic consequences, and treatment strategies. Lancet Diabetes Endocrinol 2019;7:313–24.

[5] Vinson JA, Cai Y. Nuts, especially walnuts, have both antioxidant quantity and efficacy and exhibit significant potential health benefits. Food Funct 2012;3:134–40.

[6] Becerra-Tomás N, Paz-Granier I, Kendall WCC, et al. Nut consumption and incidence of cardiovascular diseases and cardiovascular disease mortality: a meta-analysis of prospective cohort studies. Nutr Rev 2019;77:691–709.

[7] Carughii A, Bellisle F, Dougkas A, Giboreau A, Feeney MJ, Higgs J. A randomized controlled pilot study to assess effects of a daily pistachio (Pistacia Vera) afternoon snack on next-meal energy intake, satiety, and anthropometry in French women. Nutrients 2019;11:767.

[8] Liu G, Guasch-Ferré M, Hu Y, et al. Nut consumption in relation to cardiovascular disease incidence and mortality among patients with diabetes mellitus. Circ Res 2019;124:920–9.

[9] Jung JY, Park SK, Oh CM, et al. The association between metabolic syndrome and peanuts, pine nuts, almonds consumption: the Ansan and Ansong Study. Endocrin 2019;65:270–7.

[10] Chen BB, Han Y, Pan X, et al. Association between nut intake and non-alcoholic fatty liver disease risk: a retrospective case-control study in a sample of Chinese Han adults. BMJ Open 2019;9:e028961.

[11] Ma H, Xu CF, Shen Z, Yu CH, Li YM. Application of machine learning techniques for clinical predictive modeling: a cross-sectional study on nonalcoholic fatty liver disease in China. Biomed Res Int 2018;2018:304376.

[12] Yip TC, Ma AJ, Wong VW, et al. Laboratory parameter-based machine learning model for excluding non-alcoholic fatty liver disease (NAFLD) in the general population. Aliment Pharmacol Ther 2017;46:4875–56.

[13] Talliuri R, Shte S. Using the weighted area under the net benefit curve for decision curve analysis. BMC Med Inform Decis Mak 2016;16:94.

[14] Cheng L, Yan W, Zhu L, et al. Comparative analysis of IDF, ATPIII and CDS in the diagnosis of metabolic syndrome among adult inhabitants in Jiangxi Province, China. PLoS One 2017;12:e0189046.

[15] Polyzos SA, Kountouras J, Mantzoros CS. Obesity and nonalcoholic fatty liver disease: from pathophysiology to therapeutics. Metabolism 2019;92:82–97.

[16] Miyake T, Kumagi T, Hirooka M, et al. Body mass index is the most useful predictive factor for the onset of nonalcoholic fatty liver disease: a community-based retrospective longitudinal cohort study. J Gastroenterol 2013;48:413–22.

[17] VanWagner LB, Khan SS, Ning H, et al. Body mass index trajectories in young adulthood predict non-alcoholic fatty liver disease in middle age: the CARDIA cohort study. Liver Int 2018;38:706–14.

[18] Wang Y, Zeng Y, Lin C, Chen Z. Hypertension and non-alcoholic fatty liver disease proven by transient elastography. Hepatol Res 2016;46:1304–10.

[19] Donati G, Stagni B, Piscaglia F, et al. Increased prevalence of fatty liver in arterial hypertensive patients with normal liver enzymes: role of insulin resistance. Gut 2004;53:1020–3.

[20] Younossi ZM, Koenig AB, Abdellatif D, Fazely Z, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease-Meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology 2016;64:73–84.

Figure 6. The ROC curves of identified continuous variables for predicting NAFLD. The AUC for energy, MUFA, and PUFA was 0.543, 0.600, and 0.677, respectively. AUC = area under receiver operating characteristic curve, MUFA = monounsaturated fatty acids, NAFLD = non-alcoholic fatty liver disease, PUFA = polyunsaturated fatty acids, ROC = receiver operating characteristic curve.
[21] Vilar-Gomez E, Martinez-Perez Y, Calzadilla-Bertot L, et al. Weight loss through lifestyle modification significantly reduces features of nonalcoholic steatohepatitis. Gastroenterology 2015;149:367–415.

[22] European Association for the Study of the Liver (EASL), European Association for the Study of Diabetes (EASD), European Association for the Study of Obesity (EASO) EASL-EASD-EASO Clinical Practice Guidelines for the management of non-alcoholic fatty liver disease. J Hepatol 2016;64:1388–402.

[23] Lazo M, Solga SF, Horska A, et al. Effect of a 12-month intensive lifestyle intervention on hepatic steatosis in adults with type 2 diabetes. Diabetes Care 2010;33:2156–63.

[24] Patel NS, Doycheva I, Peterson MR, et al. Effect of weight loss on magnetic resonance imaging estimation of liver fat and volume in patients with nonalcoholic steatohepatitis. Clin Gastroenterol Hepatol 2015;13:561.e1–8.e1.

[25] Mansour-Ghanaei F, Hadi A, Pourmasoumi M, Joukar F, Golpour S, Najafgholizadeh A. Green tea as a safe alternative approach for nonalcoholic fatty liver treatment: a systematic review and meta-analysis of clinical trials. Phytother Res 2018;32:1876–84.

[26] Koo SI, Noh SK. Green tea as inhibitor of the intestinal absorption of lipids: potential mechanism for its lipid-lowering effect. J Nutr Biochem 2007;18:179–83.

[27] Tipoe GL, Leung TM, Hung MW, Fung ML. Green tea polyphenols as an anti-oxidant and anti-inflammatory agent for cardiovascular protection. Cardiovasc Hematol Disord Drug Targets 2007;7:135–44.

[28] Lee MS, Kim CT, Kim Y. Green tea (-)-epigallocatechin-3-gallate reduces body weight with regulation of multiple genes expression in adipose tissue of diet-induced obese mice. Ann Nutr Metab 2009;54:151–7.

[29] Kim Y, Keogh JB, Clifton PM. Benefits of nut consumption on insulin resistance and cardiovascular risk factors: multiple potential mechanisms of actions. Nutrients 2017;9:1271.

[30] Han JM, Jo AN, Lee SM, et al. Associations between intakes of individual nutrients or whole food groups and non-alcoholic fatty liver disease among Korean adults. J Gastroenterol Hepatol 2014;29:1265–72.

[31] George ES, Forsyth A, Itriopoulos C, et al. Practical dietary recommendations for the prevention and management of nonalcoholic fatty liver disease in adults. Adv Nutr 2018;9:30–40.

[32] Alper CM, Mattes RD. Effects of chronic peanut consumption on energy balance and hedonics. Int J Obes Relat Metab Disord 2002;26:1129–37.