Hepatology International (2022) 16:846–857
https://doi.org/10.1007/s12072-022-10368-x

ORIGINAL ARTICLE

Associations between intake of starchy and non-starchy vegetables and risk of hepatic steatosis and fibrosis

Xiude Li[1,2,3,4] · **Tengfei Zhang**[1] · **Haowei Li**[1] · **Zhihao Zhou**[1] · **Meiling Li**[1] · **Xueke Zeng**[1] · **Hu Yang**[1] · **Mingyi Zhang**[1] · **Yong Huang**[1] · **Yu Zhu**[1] · **Zhuang Zhang**[1] · **Yanan Ma**[5] · **Wanshui Yang**[1,2,3,4]

Received: 16 March 2022 / Accepted: 13 May 2022 / Published online: 21 June 2022

Abstract

Background Current dietary guidelines generally treat all types of vegetables the same. However, whether specific vegetables are more beneficial or deleterious for preventing chronic liver disease (CLD) remains uncertain.

Methods We investigated the associations between starchy and non-starchy vegetables and the odds of hepatic steatosis and fibrosis in a US nationwide cross-sectional study. Diet was assessed by the 24-h dietary recalls. Hepatic steatosis and fibrosis were defined based on vibration-controlled transient elastography (TE). Multiple logistic regression was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs).

Results Among 4170 participants with reliable TE test, 1436 were diagnosed with steatosis, 255 with advanced fibrosis. Increased intake of total starchy vegetables was associated with higher odds of steatosis (OR per 1-SD increment 1.11, 95% CI 1.01–1.24) and advanced fibrosis (OR = 1.39, 95% CI 1.15–1.69). Similar positive associations were observed for potatoes. Conversely, intakes of total non-starchy (OR = 0.82, 95% CI 0.71–0.95) and dark-green vegetables (OR = 0.89, 95% CI 0.82–0.97) were inversely associated with steatosis prevalence. Replacing 5% of energy from starchy vegetables (OR = 0.65, 95% CI 0.44–0.97) or potatoes (OR = 0.65, 95% CI 0.43–0.97) with equivalent energy from dark-green vegetables was associated with lower odds of steatosis.

Conclusions These findings support the recommendation to limit starchy vegetable intake and increase non-starchy vegetable intake in CLD prevention, and provide evidence for the potential health benefit from dietary substitution of non-starchy vegetables for starchy vegetables.

Keywords Controlled attenuation parameter · Chronic liver disease · Dark-green vegetables · Inflammation · Liver stiffness measurement · Nonalcoholic fatty liver disease · Potatoes · Substitution analysis · Transient elastography · United States

Abbreviations

BMI	Body mass index
CAP	Controlled attenuation parameter
CI	Confidence interval
CLD	Chronic liver disease
CRP	C-reactive protein
GI	Glycemic index
GL	Glycemic load
HbA1c	Hemoglobin A1c
HBV	Hepatitis B virus
HCV	Hepatitis C virus
hs-CRP	High-sensitivity CRP
IL-6	Interleukin 6
IQR	Interquartile range
LSM	Liver stiffness measurement
MEC	Mobile Examination Center
METS	Metabolic equivalent tasks
NAFLD	Nonalcoholic fatty liver disease
NCHS	National Centers for Health Statistics
NHANES	National Health and Nutrition Examination Survey
OR	Odds ratio

Xiude Li, Tengfei Zhang and Haowei Li have contributed equally as co-first authors.

Wanshui Yang and Yanan Ma have contributed equally as co-senior authors.

✉ Yanan Ma
ynma@cmu.edu.cn

✉ Wanshui Yang
wanshuiyang@gmail.com

Extended author information available on the last page of the article

SD	Standard deviation
TE	Transient elastography

Introduction

Nonalcoholic fatty liver disease (NAFLD) affects approximately 25% of global population, and can proceed to more severe liver diseases such as hepatic cirrhosis or liver cancer. There are no approved drugs for treating such diseases. Thus, lifestyle changes, including dietary modifications, to achieve weight loss, are key to prevent and reduce the progression of NAFLD. Recently, Mediterranean diet, characterized by high intake of vegetables, and the combination of physical exercise, are highly proposed for preventing and managing NAFLD.

A growing body of evidence, however, showed that specific vegetables may have heterogeneous health impacts. For example, higher intake of starchy vegetables, such as potatoes, peas, and corn, were associated with greater weight gain [1]. Also, starchy vegetables might not confer similar health benefits to non-starchy vegetables on multiple health outcomes such as mortality [2], which could be partly due to their high glycemic load (GL) [3] and antioxidant loss during processing [4]. In addition, previous observational studies of vegetable intake and NAFLD are limited with mixed results [5–7], and did not distinguish between starchy and non-starchy vegetables. Likewise, current dietary guidelines generally treat all types of vegetables equally [8, 9], highlighting the importance to investigate potentially distinct health effects of different subgroups of vegetables. To the best of our knowledge, no studies to date have comprehensively examined whether intake of specific vegetables might be more beneficial or deleterious for preventing chronic liver disease (CLD). Therefore, we hypothesized that starchy vegetables might be positively, while non-starchy vegetables might be inversely associated with hepatic steatosis or fibrosis.

To test this hypothesis, we assessed the association between specific vegetables (i.e., starchy and non-starchy vegetables and their subgroups) and the prevalence of hepatic steatosis and fibrosis, with the use of a nationwide cross-sectional data from the US National Health and Nutrition Examination Survey (NHANES). To facilitate translations of dietary recommendations, we also conducted substitution analysis to investigate association between the replacement of starchy vegetables with non-starchy vegetables and the odds of steatosis and fibrosis.

Methods

Study population

Participants in the present study were selected from the 2017 to 2018 cycle of the NHANES, in which transient elastography (TE) examination was performed for the first time in the survey. NHANES is a continuous, cross-sectional survey conducted by the National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention. Every year, a nationally representative sample of approximately 5000 persons of all ages is investigated. Details of the NHANES design and data collection methods have been described previously [10]. Written informed consent is obtained from all participants. The NCHS Research Ethics Review Board approved the NHANES study protocols (Protocol #2011–17; Protocol #2018–01). The NHANES data are publicly available at https://www.cdc.gov/nchs/nhanes/index.htm.

Participants aged ≥ 18 years were included in this analysis. In addition, we excluded individuals if they had missing dietary data ($n = 873$) or implausible energy intake (< 600 or > 3500 kcal/day for female and < 800 or > 4200 kcal/day for male, $n = 221$), or did not receive TE examination ($n = 70$), or had unreliable TE data ($n = 522$). A total of 4170 eligible participants (2133 female and 2037 male) were included in the analysis (Supplementary Fig. 1).

Dietary assessment

Diet was measured using 24-h dietary recall by trained dietary interviewers. Multiple-pass approaches were used to enhance complete and accurate food recall and decrease the respondent burden. From 1999 to 2002, only single dietary interview was administered in person at the NHANES Mobile Examination Center (MEC). Since 2003, a second interview was performed by telephone 3–10 days after the first recall. In the current analysis, most participants ($n = 3662$, 87.8%) had two valid 24-h dietary recalls.

Definition of vegetables was based on the US Department of Agriculture Food and Nutrition Database for Dietary Studies (Supplementary Table 1). Starchy vegetables included white potatoes (e.g., baked, boiled, mashed, scalloped, and fried potatoes and potato chips) and other starchy vegetables (e.g., immature peas, lima beans, and corn). Non-starchy vegetables included dark-green vegetables (e.g., raw and cooked broccoli, romaine, and collards), deep-yellow vegetables (e.g., carrots, pumpkin, and winter squash), and other non-starchy vegetables (e.g., tomatoes and lettuce).

Assessments of covariates

Sex, age, race/ethnicity, education, marital status, income, physical activity, and smoking were collected during household interviews using standardized questionnaires. Height, body weight, and alcohol consumption data were assessed in the MEC. The ratio of family income to poverty was used to measure family income. We used metabolic equivalent tasks (METS)-hours/week to estimate the amount of physical activity. Body mass index (BMI) was calculated as the weight in kilograms divided by the square of height in meters (kg/m^2). Hepatitis B virus (HBV) infection was determined by the presence of hepatitis B surface antigen, while hepatitis C antibody or ribonucleic acid being positive indicate hepatitis C virus (HCV) infection. Diabetes was defined as self-reported diagnosis of diabetes, fasting glucose level ≥ 126 mg/dL, or a hemoglobin A1c (HbA1c) level ≥ 6.5%. Alcohol drinking status including never drinking, ever drinking, light drinking, moderate drinking, and heavy drinking was defined according to the method by the US Centers for Disease Control and Prevention [11].

Ascertainments of hepatic steatosis and fibrosis

Vibration-controlled TE was conducted in the MEC by trained health technicians, using the FibroScan® model 502 V2 Touch equipped with a medium or extra-large probe. Consistent with previous studies [10, 12], we used TE-derived controlled attenuation parameter (CAP) to define S1, S2, and S3 hepatic steatosis, with cut-off values of median CAP ≥ 274, 290, and 302 dB/m, respectively. Simultaneously, liver stiffness measurement (LSM) was derived to define significant fibrosis, advanced fibrosis, and cirrhosis, with cut-off values of median LSM ≥ 8.2, 9.7, and 13.6 kPa, respectively [10, 12]. TE examinations were considered as valid only if more than 10 LSMs were obtained after a fasting time no less than 3 h, with an interquartile range (IQR) to median ratio less than 30%. By comparing FibroScan® for the detection of steatosis and fibrosis against biopsy, the area under the receiver operating characteristic curves ranged from 0.70 to 0.89 among patients with NAFLD, and probe type and steatosis seemed not to affect LSM [12].

Statistical analysis

All analyses incorporated appropriate sampling weights, stratification, and clustering of the complex sampling design in the present study to ensure nationally representative estimates. The prevalences of hepatic steatosis and fibrosis were standardized by 2020 US Census population [10]. Consistent with previous study [2], we defined the standard serving size as 80 g for all types of vegetables, and categorized vegetable intake into 3 groups (i.e., 0, 0–1, and ≥ 1 servings/day). The multivariable logistic regression model was used to calculate the odds ratios (ORs) and 95% confidence intervals (CIs). We also presented the ORs per 1-standard deviation (SD) increment of each vegetable intake. Model 1 was adjusted for age. Model 2 was further adjusted for sex, total energy intake, race/ethnicity, education, marital status, ratio of family income to poverty, physical activity, smoking, alcohol consumption, diabetes, HBV, HCV, BMI, and intake of whole grains, refined grains, fruits, nuts, tea, coffee, fruit juices, and sugar sweetened beverages. Selection of these covariates was based on observed incomparability of characteristics of participants (Table 1) and the previously identified risk factors for the outcomes. A missing value indicator was created for each covariate in the models if possible. Linear trend was tested by treating each exposure as a continuous variable in the models. We used restricted cubic spline to evaluate the potential non-linear relationships between vegetable consumption and odds of hepatic steatosis and fibrosis.

Intakes of total starchy vegetables and total non-starchy vegetables were dichotomized into "no" (i.e., no consumption) and "yes" (i.e., > 0 servings/day), resulting in four mutually exclusive groups (yes–no, no–no, yes–yes, and no–yes). These four groups were then evaluated in relation to the levels of plasma markers of glucose metabolism, lipid metabolism, and inflammation markers. The results of the multivariate linear regression analysis were presented as the percentage differences in markers comparing starchy and non-starchy vegetable consumers in different categories to the reference group [i.e., non-starchy vegetables (yes)–starchy vegetables (no)], expressed as [exp(β) – 1] × 100%. All marker concentrations were transformed using the natural logarithm to improve normality.

We used leave-one-out model [13] to assess the associations between replacement of 5% of energy from starchy vegetables with equivalent amounts of energy from non-starchy vegetables and odds of hepatic steatosis and fibrosis. In sensitivity analysis, we repeated analysis using different cut-off values to define hepatic steatosis (CAP ≥ 288 dB/m for any steatosis) [14] and fibrosis (LSM ≥ 10.1 kPa for advanced fibrosis) [15]. We also repeated analysis within persons who did not have hepatitis B and/or C. In subgroup analysis, we stratified analyses by age, sex, race/ethnicity, marital status, education level, ratio of family income to poverty, smoking status, alcohol consumption, physical activity, BMI, or diabetes. We used Wald test to examine whether interaction terms between these variables and exposures were statistically significant. All statistical analyses were performed using SAS version 9.4 (SAS Institute Inc, Cary, NC).

Results

Characteristics of participants

Of the 4170 participants (mean age 49.4 years; SD 18.3 years), 1436 (age-standardized prevalence 33.8%) were diagnosed with hepatic steatosis (CAP ≥ 290 dB/m), and 255 (5.6%) with advanced fibrosis (LSM ≥ 9.7 kPa). Potato and other non-starchy vegetable intakes contribute substantially to total vegetable intake, with proportions

Table 1 Age-adjusted characteristics of participants according to individual vegetable intake in NHANES (2017–2018)

	Total starchy vegetables (servings/day)			Total non-starchy vegetables (servings/day)		
	0	< 1	≥ 1	0	< 1	≥ 1
Age, years	49.1 (18.1)	48.5 (18.2)	51.3 (18.5)	45.7 (18.7)	48.4 (18.7)	51.9 (17.3)
Female, %	50.6	54.6	47.4	44.7	50.5	54.9
BMI, kg/m^2	29.0 (6.7)	29.7 (7.2)	29.7 (7.4)	29.5 (7.3)	29.8 (7.1)	29.0 (6.8)
Total energy, kcal/d	1855 (677)	1995 (706)	2221 (740)	1891 (735)	1973 (712)	2037 (706)
Diabetes, %	18.8	18.6	19.9	19.7	20.3	17.5
HBV infection, %	0.5	0.5	0.2	0.4	0.5	0.4
HCV infection, %	1.8	1.9	1.5	3.0	2.1	1.1
Race/ethnicity, %						
Non-Hispanic white	32.3	33.1	42.4	35.7	35.7	34.1
Non-Hispanic black	17.8	27.0	26.5	25.7	24.0	20.2
Other races	49.9	39.9	31.1	38.5	40.3	45.7
Education, %						
≤ 12th grade	21.3	16.1	17.2	25.6	20.3	14.0
High school graduate	23.4	25.6	26.3	28.9	26.6	21.1
More than high school	55.1	58.2	56.4	45.3	53.0	64.8
Marital status, %						
Married	56.3	56.4	57.6	51.1	56.9	59.6
Widowed/divorced/separated	22.5	20.1	19.9	22.9	20.7	20.6
Never married	16.3	18.1	17.6	19.7	17.4	15.8
Ratio of family income to poverty						
< 1.30	25.3	25.0	25.2	32.9	25.2	21.5
1.30–3.49	34.0	36.0	37.7	35.4	37.3	34.0
≥ 3.50	28.8	27.4	26.4	18.6	25.2	34.1
Physical activity, METS-h/week						
< 8.3	33.2	33.8	38.3	37.2	35.6	32.8
8.3–16.7	10.5	10.1	7.2	8.7	9.3	10.1
> 16.7	55.7	55.4	53.2	52.7	54.1	56.6
Smoking, %						
Never smokers	60.3	59.8	57.7	50.0	58.0	64.7
Former smokers	23.1	23.5	24.5	23.8	24.6	22.9
Current smokers	16.5	16.7	17.8	26.1	17.4	12.4
Alcohol consumption, %						
Never drinkers	12.3	9.8	8.6	10.6	10.5	10.6
Former drinkers	18.5	20.0	20.9	24.2	19.6	17.7
Light drinkers	47.4	50.3	48.3	46.6	48.3	49.5
Moderate drinkers	13.6	12.6	14.2	10.9	13.1	14.9
Heavier drinkers	5.3	2.5	5.5	5.3	6.1	4.2

Values were presented as means (SD) or percentages. All variables were standardized to the age distribution of the study population except for age. Of note, the summing proportions for some categories are not 100% due to missing values or rounding

BMI body mass index, *HBV* hepatitis B virus, *HCV* hepatitis C virus, *METS* metabolic equivalent tasks, *NHANES* National Health and Nutrition Examination Survey, *SD* standard deviation

of energy of 57% from white potatoes, 29% from other non-starchy vegetables, 6% from other starchy vegetables, and both 4% from dark-green and deep-yellow vegetables (Supplementary Fig. 2). Participants with higher consumption of starchy vegetables were more likely to be non-Hispanic white and current smokers, had lower family income to poverty ratio, and were less physically active. These trends were reversed for the non-starchy vegetables (Table 1).

Vegetables and steatosis and fibrosis

Compared with no consumption, daily consumption of one serving of total starchy vegetables or more was associated higher prevalence of steatosis and advanced fibrosis with ORs of 1.42 (95% CI 1.15–1.76, $p_{\text{trend}} = 0.035$) and 1.68 (95% CI 1.06–2.68, $p_{\text{trend}} = 0.001$), respectively (Table 2). For the same comparison, higher consumption of potatoes was associated with higher odds of steatosis (OR = 1.30, 95% CI 1.03–1.64, $p_{\text{trend}} = 0.043$) and fibrosis (OR = 1.72, 95% CI 1.16–2.54, $p_{\text{trend}} = 0.001$). Other starchy vegetable intake was not associated with steatosis or fibrosis. Restricted cubic spline did not support a non-linear association between starchy vegetables and odds of hepatic steatosis or fibrosis (all $p_{\text{non-linearity}} > 0.05$, Fig. 1).

Table 2 Association between starchy vegetable intake and hepatic steatosis and fibrosis in NHANES (2017–2018)

	OR (95% CI)				p_{trend}[c]
	0 servings/day	< 1 serving/day	≥ 1 serving/day	Per 1-SD increase	
Hepatic steatosis (CAP ≥ 290 dB/m)					
Total starchy vegetables					
No. of cases/participants	576/1804	487/1390	373/976		
Model 1[a]	Reference	1.12 (0.91–1.38)	1.37 (1.10–1.71)	1.12 (1.03–1.22)	0.008
Model 2[b]	Reference	1.10 (0.84–1.43)	1.42 (1.15–1.76)	1.11 (1.01–1.24)	0.035
Potatoes					
No. of cases/participants	654/2020	463/1333	319/817		
Model 1[a]	Reference	1.16 (0.93–1.43)	1.37 (1.13–1.68)	1.12 (1.04–1.22)	0.005
Model 2[b]	Reference	1.09 (0.87–1.37)	1.30 (1.03–1.64)	1.11 (1.00–1.24)	0.043
Other starchy vegetables					
No. of cases/participants	1250/3672	134/344	52/154		
Model 1[a]	Reference	1.15 (0.83–1.60)	0.76 (0.47–1.21)	1.03 (0.94–1.12)	0.557
Model 2[b]	Reference	1.35 (1.00–1.84)	0.83 (0.44–1.57)	1.02 (0.92–1.13)	0.686
Hepatic fibrosis (LSM ≥ 9.7 kPa)					
Total starchy vegetables					
No. of cases	112	73	70		
Model 1[a]	Reference	0.93 (0.65–1.33)	2.02 (1.21–3.35)	1.40 (1.14–1.72)	0.001
Model 2[b]	Reference	0.80 (0.58–1.12)	1.68 (1.06–2.68)	1.39 (1.15–1.69)	0.001
Potatoes					
No. of cases	125	70	60		
Model 1[a]	Reference	0.93 (0.59–1.49)	2.00 (1.29–3.10)	1.41 (1.15–1.74)	0.001
Model 2[b]	Reference	0.78 (0.52–1.18)	1.72 (1.16–2.54)	1.42 (1.16–1.73)	0.001
Other starchy vegetables					
No. of cases	227	18	10		
Model 1[a]	Reference	0.99 (0.43–2.28)	0.86 (0.35–2.09)	1.04 (0.84–1.27)	0.773
Model 2[b]	Reference	0.71 (0.28–1.82)	0.94 (0.35–2.52)	0.98 (0.74–1.29)	0.885

BMI body mass index, *CAP* controlled attenuation parameter, *CI* confidence interval, *HBV* hepatitis B virus, *HCV* hepatitis C virus, *LSM* liver stiffness measurement, *METS* metabolic equivalent tasks, *NHANES* National Health and Nutrition Examination Survey; OR, odds ratio, *SD* standard deviation

[a] Model 1 was adjusted for age (18–29, 30–39, 40–49, 50–59, or ≥ 60 years)

[b] Model 2 was further adjusted for sex (male, female), total energy intake (kcal/day, tertile), race/ethnicity (non-Hispanic white, non-Hispanic black, or other races), education (≤ 12th grade, high school graduate, or more than high school), marital status (married, widowed/divorced/separated, or never married), ratio of family income to poverty (< 1.30, 1.30–3.49, or ≥ 3.50), physical activity (< 8.3, 8.3–16.7, or > 16.7 METS-h/week), smoking (never smokers, former smokers, or current smokers), alcohol consumption (never drinkers, former drinkers, light drinkers, moderate drinkers, or heavier drinkers), diabetes (no, yes), HBV infection (no, yes), HCV infection (no, yes), BMI (< 18.5, 18.5–24.9, 25.0–29.9, or ≥ 30.0 kg/m^2), whole grains (continuous, g/day), refined grains (continuous, g/day), fruits (continuous, g/day), nuts (continuous, g/day), tea (continuous, g/day), coffee (continuous, g/day), fruit juices (continuous, g/day), sugar sweetened beverages (continuous, g/day), and non-starchy vegetables (0, < 1, or ≥ 1 servings/day). Of note, potatoes and other starchy vegetables were mutually adjusted

[c] Linear trend test was conducted by treating the intake of vegetables as a continuous variable in the models

Participants with higher intake of total non-starchy vegetables (OR = 0.73, 95% CI 0.49–1.06, $p_{\text{trend}} = 0.008$), or dark-green vegetables (OR = 0.63, 95% CI 0.40–1.00, $p_{\text{trend}} = 0.010$) had lower odds of steatosis (Table 3). However, deep-yellow vegetables ($p_{\text{trend}} = 0.984$) and other non-starchy vegetables ($p_{\text{trend}} = 0.052$) did not show any significant associations with steatosis. A non-significant association between total or any subgroups of non-starchy vegetable intake and prevalence of fibrosis was found.

Replacing 5% of energy from total starchy vegetables (OR = 0.65, 95% CI 0.44–0.97) or potatoes (OR = 0.65, 95% CI 0.43–0.97) with dark-green vegetables was associated with lower odds of steatosis (Fig. 2). However, substituting total or any specific non-starchy vegetable for starchy vegetables was not associated with prevalence of fibrosis (data not shown). Compared with those who consumed non-starchy vegetables but not starchy vegetables, participants consumed starchy vegetables but not non-starchy vegetables had higher high-sensitivity C-reactive protein (hs-CRP) and lower total cholesterol. No significant differences were observed for other markers (Supplementary Table 2).

Sensitivity and subgroup analysis

In sensitivity analysis, the results were not appreciably changed when using different cut-off values to define steatosis and advanced fibrosis (Supplementary Table 3), or restricting analysis within persons who did not have hepatitis B and/or C (Fig. 3 and Supplementary Fig. 3). In subgroup analysis, no differential associations between each specific vegetable intake and odds of liver diseases were found according to age, sex, race/ethnicity, education level, marital status, ratio of family income to poverty, smoking status, alcohol drinking, physical activity, BMI, or diabetes (Fig. 3 and Supplementary Fig. 3).

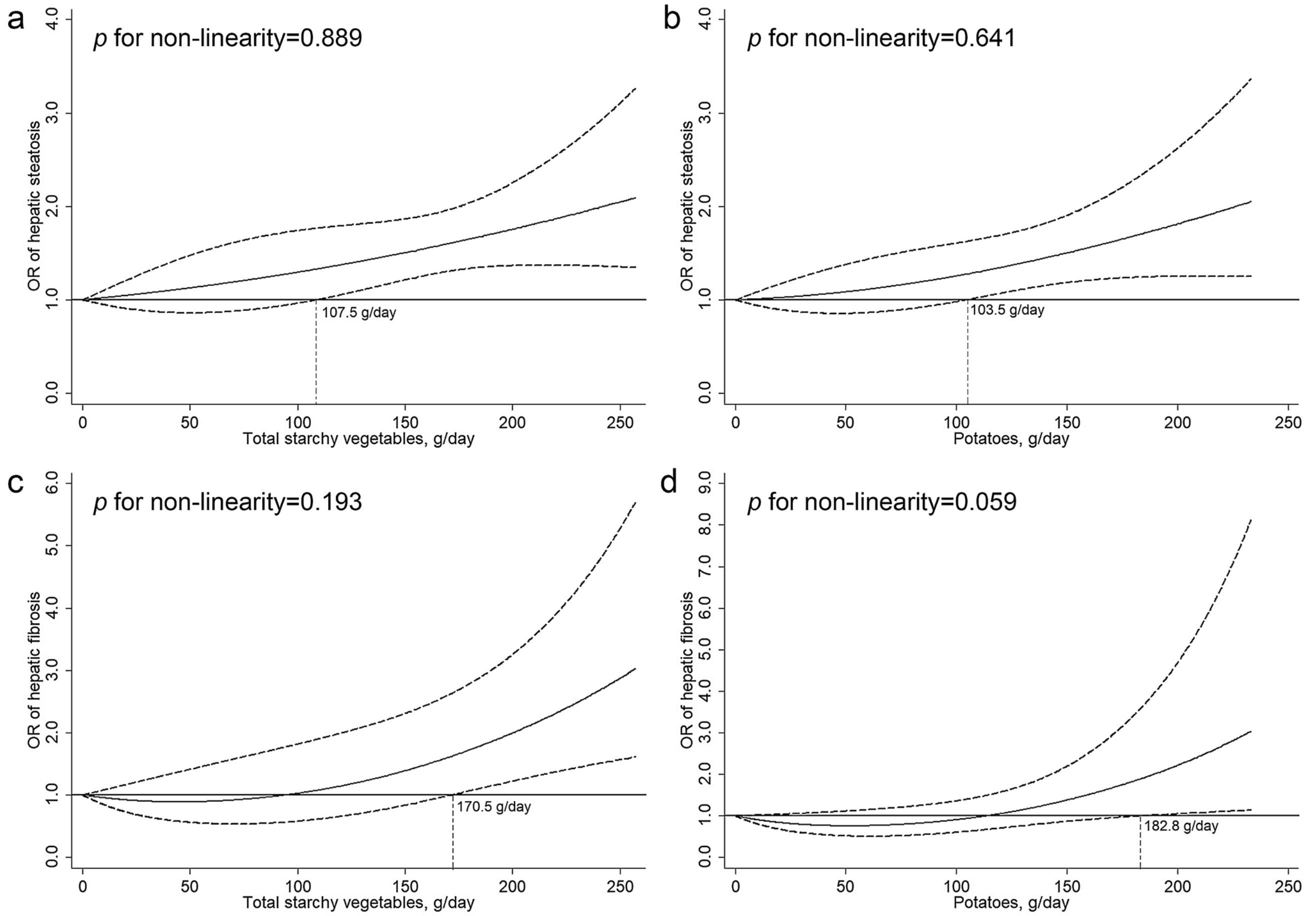


Fig. 1 Dose–response relationship between starchy vegetable consumption and hepatic steatosis (CAP ≥ 290 dB/m) (**a**, **b**) and fibrosis (LSM ≥ 9.7 kPa) (**c**, **d**) in NHANES (2017–2018). *CAP* controlled attenuation parameter, *CI* confidence interval, *LSM* liver stiffness measurement, *NHANES* National Health and Nutrition Examination Survey, *OR* odds ratio. Covariates adjusted in the models were the same as those in model 2 in Table 2 (see footnote in Table 2). Solid lines indicate estimates, and dashed lines depict 95% CI

Table 3 Association between non-starchy vegetable intake and hepatic steatosis and fibrosis in NHANES (2017–2018)

	OR (95%CI)				p_{trend}[c]
	0 servings/day	< 1 serving/day	≥ 1 serving/day	Per 1-SD increase	
Hepatic steatosis (CAP ≥ 290 dB/m)					
Total non-starchy vegetables					
No. of cases/participants	249/782	578/1586	609/1802		
Model 1[a]	Reference	1.13 (0.79–1.61)	0.78 (0.54–1.12)	0.80 (0.70–0.91)	< 0.001
Model 2[b]	Reference	0.86 (0.60–1.24)	0.73 (0.49–1.06)	0.82 (0.71–0.95)	0.008
Dark-green vegetables					
No. of cases/participants	1171/3289	196/627	69/254		
Model 1[a]	Reference	0.68 (0.51–0.91)	0.61 (0.43–0.90)	0.87 (0.77–0.99)	0.032
Model 2[b]	Reference	0.89 (0.59–1.36)	0.63 (0.40–1.00)	0.89 (0.82–0.97)	0.010
Deep-yellow vegetables					
No. of cases/participants	1211/3485	179/559	46/126		
Model 1[a]	Reference	0.65 (0.47–0.90)	0.87 (0.50–1.50)	0.93 (0.83–1.04)	0.175
Model 2[b]	Reference	0.90 (0.60–1.33)	1.28 (0.61–2.71)	1.00 (0.86–1.16)	0.984
Other non-starchy vegetables					
No. of cases/participants	302/932	625/1768	509/1470		
Model 1[a]	Reference	1.06 (0.79–1.42)	0.75 (0.51–1.10)	0.82 (0.71–0.94)	0.005
Model 2[b]	Reference	0.84 (0.61–1.17)	0.74 (0.45–1.20)	0.85 (0.72–1.00)	0.052
Hepatic fibrosis (LSM ≥ 9.7 kPa)					
Total non-starchy vegetables					
No. of cases	40	104	111		
Model 1[a]	Reference	1.75 (1.13–2.72)	1.78 (1.12–2.81)	0.92 (0.77–1.10)	0.361
Model 2[b]	Reference	1.51 (0.99–2.28)	1.89 (1.13–3.14)	1.02 (0.86–1.21)	0.825
Dark-green vegetables					
No. of cases	195	39	21		
Model 1[a]	Reference	0.90 (0.50–1.64)	1.37 (0.65–2.93)	1.12 (0.97–1.28)	0.126
Model 2[b]	Reference	1.42 (0.80–2.53)	1.52 (0.68–3.39)	1.13 (1.00–1.27)	0.053
Deep-yellow vegetables					
No. of cases	215	34	6		
Model 1[a]	Reference	0.52 (0.30–0.90)	1.49 (0.43–5.12)	1.05 (0.82–1.35)	0.677
Model 2[b]	Reference	0.77 (0.41–1.44)	2.31 (0.71–7.53)	1.14 (0.93–1.40)	0.211
Other non-starchy vegetables					
No. of cases	52	116	87		
Model 1[a]	Reference	1.55 (1.03–2.34)	1.38 (0.85–2.23)	0.82 (0.66–1.01)	0.059
Model 2[b]	Reference	1.30 (0.87–1.95)	1.46 (0.95–2.27)	0.86 (0.65–1.15)	0.305

BMI body mass index, *CAP* controlled attenuation parameter, *CI* confidence interval, *HBV* hepatitis B virus, *HCV* hepatitis C virus, *LSM* liver stiffness measurement, *METS* metabolic equivalent tasks, *NHANES* National Health and Nutrition Examination Survey, *OR* odds ratio, *SD* standard deviation

[a] Model 1 was adjusted for age (18–29, 30–39, 40–49, 50–59, or ≥ 60 years)

[b] Model 2 was further adjusted for sex (male, female), total energy intake (kcal/day, tertile), race/ethnicity (non-Hispanic white, non-Hispanic black, or other races), education (≤ 12th grade, high school graduate, or more than high school), marital status (married, widowed/divorced/separated, or never married), ratio of family income to poverty (< 1.30, 1.30–3.49, or ≥ 3.50), physical activity (< 8.3, 8.3–16.7, or > 16.7 METS-h/week), smoking (never smokers, former smokers, or current smokers), alcohol consumption (never drinkers, former drinkers, light drinkers, moderate drinkers, or heavier drinkers), diabetes (no, yes), HBV infection (no, yes), HCV infection (no, yes), BMI (< 18.5, 18.5–24.9, 25.0–29.9, or ≥ 30.0 kg/m^2), whole grains (continuous, g/day), refined grains (continuous, g/day), fruits (continuous, g/day), nuts (continuous, g/day), tea (continuous, g/day), coffee (continuous, g/day), fruit juices (continuous, g/day), sugar sweetened beverages (continuous, g/day), and starchy vegetables (0, < 1, or ≥ 1 servings/day). Of note, dark-green vegetables, deep-yellow vegetables, and other non-starchy vegetables were mutually adjusted

[c] Linear trend test was conducted by treating the intake of vegetables as a continuous variable in the models

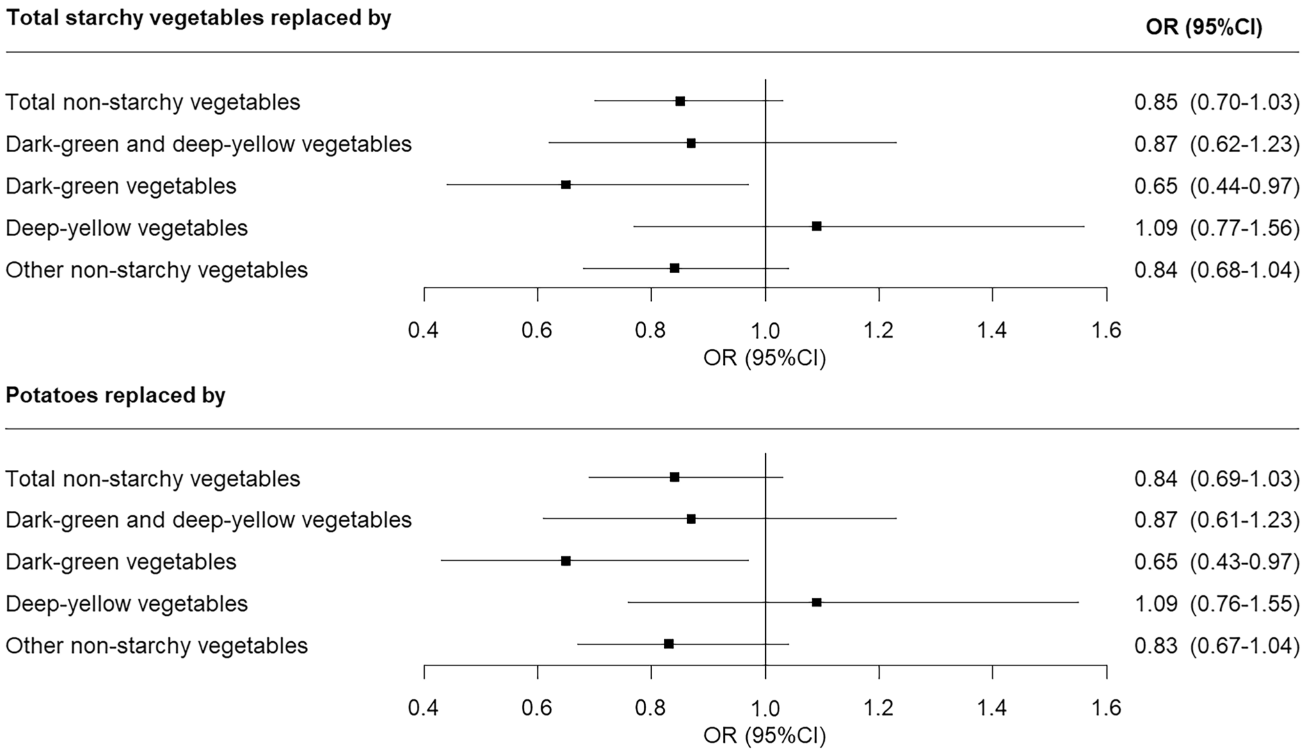


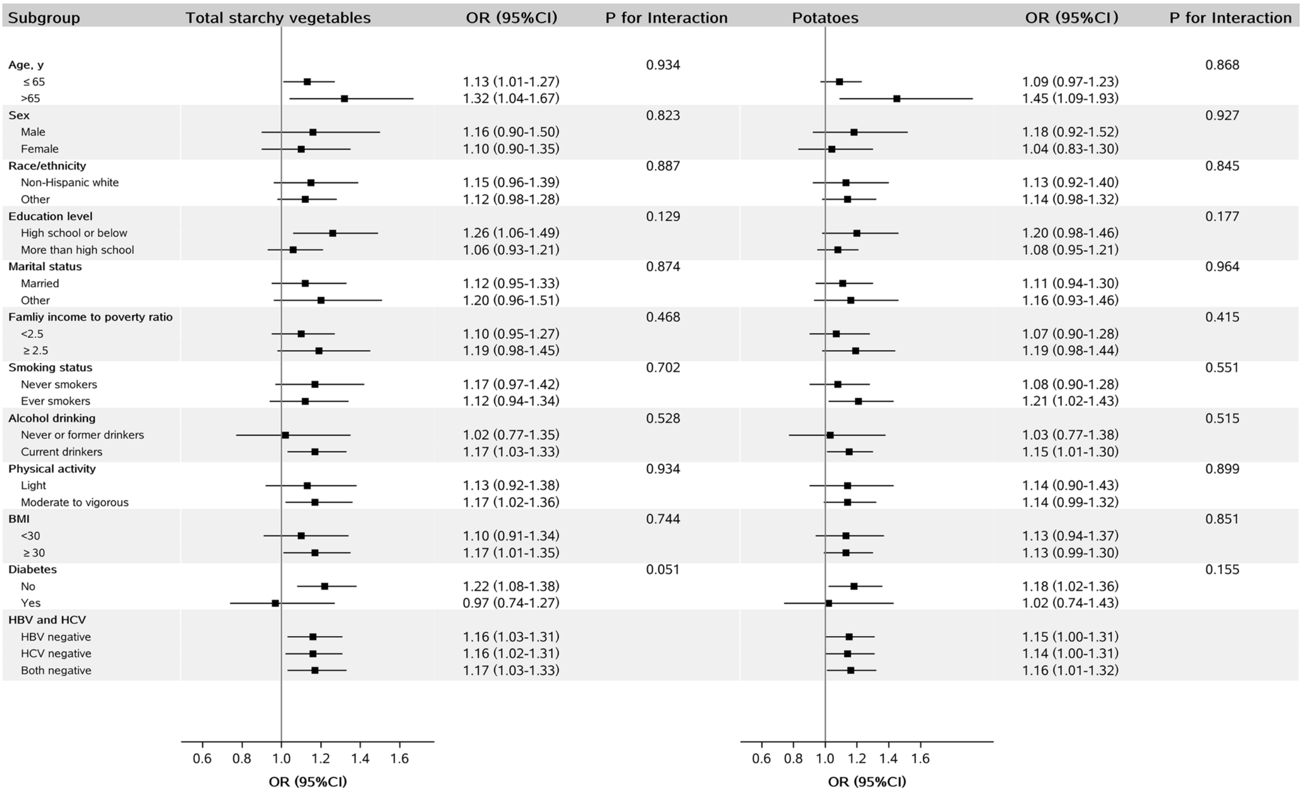
Fig. 2 Association between replacement of 5% of energy from total starchy vegetables and potatoes with equivalent amount of energy from non-starchy vegetables and odds of hepatic steatosis (CAP ≥ 290 dB/m) in NHANES (2017–2018). *CAP* controlled attenuation parameter, *CI* confidence interval, *NHANES* National Health and Nutrition Examination Survey, *OR* odds ratio. Covariates adjusted in the models were the same as those in model 2 in Table 2 (see footnote in Table 2)

Discussion

In this nationwide cross-sectional study, we generally showed that starchy and non-starchy vegetables had an opposite association with prevalence of hepatic steatosis and fibrosis. Specially, intake of total starchy vegetables or potatoes was associated with high odds of hepatic steatosis and advanced fibrosis. Conversely, higher intake of total non-starchy or dark-green vegetables was associated with lower odds of steatosis among US adults. In addition, substituting 5% of energy from dark-green vegetables for total starchy vegetables or potatoes was associated with reduced odds of hepatic steatosis. These findings did not support the current dietary guidelines [8, 9] and federal nutrition assistance programs' positions on treating all types of vegetables equally without considering their potentially differential nutritional properties and health effects.

Observational studies of vegetables and CLD risk are limited and have yielded inconsistent results. A prospective cohort study among Korean adults showed that total vegetable intake was associated with a lower risk of NAFLD [7], whereas a cross-sectional study in Japan found no association between total vegetables and odds of NAFLD [5]. This discrepancy could be partly due to the different study design (cohort vs. cross-sectional study). Among two case–control studies in China, one reported a lower [6] and another reported a higher [16] odds of NAFLD associated with potato intake. A possible reason for this discrepancy could be due to differences in study populations (faculty members and staff vs. participants who received annual physical examinations). Among three existing studies on the association between non-starchy vegetables and NAFLD, two reported an inverse [17, 18] and one reported a non-significant [5] association. In addition, in a cross-sectional study of overweight Latino youth, higher non-starchy vegetable intake was associated with lower liver fat deposition [19], which was generally consistent with our findings.

Our results are biologically plausible. First, starchy vegetables generally have higher glycemic index (GI), and may thereby raise glucose levels faster than non-starchy vegetables, and lead to increased insulin response [20]. Consistently, we observed higher levels of fasting glucose and HbA1c among participant who consumed starchy vegetables than among those who consumed non-starchy vegetables, despite no statistical significance. Previous studies showed that diets or foods with high GI or GL levels were associated with hepatic steatosis and/or fibrosis [10, 21–23]. Also, in our previous study, higher dietary insulinemic potential was



Subgroup	Total starchy vegetables OR (95%CI)	P for Interaction	Potatoes OR (95%CI)	P for Interaction
Age, y		0.934		0.868
≤65	1.13 (1.01-1.27)		1.09 (0.97-1.23)	
>65	1.32 (1.04-1.67)		1.45 (1.09-1.93)	
Sex		0.823		0.927
Male	1.16 (0.90-1.50)		1.18 (0.92-1.52)	
Female	1.10 (0.90-1.35)		1.04 (0.83-1.30)	
Race/ethnicity		0.887		0.845
Non-Hispanic white	1.15 (0.96-1.39)		1.13 (0.92-1.40)	
Other	1.12 (0.98-1.28)		1.14 (0.98-1.32)	
Education level		0.129		0.177
High school or below	1.26 (1.06-1.49)		1.20 (0.98-1.46)	
More than high school	1.06 (0.93-1.21)		1.08 (0.95-1.21)	
Marital status		0.874		0.964
Married	1.12 (0.95-1.33)		1.11 (0.94-1.30)	
Other	1.20 (0.96-1.51)		1.16 (0.93-1.46)	
Famliy income to poverty ratio		0.468		0.415
<2.5	1.10 (0.95-1.27)		1.07 (0.90-1.28)	
≥2.5	1.19 (0.98-1.45)		1.19 (0.98-1.44)	
Smoking status		0.702		0.551
Never smokers	1.17 (0.97-1.42)		1.08 (0.90-1.28)	
Ever smokers	1.12 (0.94-1.34)		1.21 (1.02-1.43)	
Alcohol drinking		0.528		0.515
Never or former drinkers	1.02 (0.77-1.35)		1.03 (0.77-1.38)	
Current drinkers	1.17 (1.03-1.33)		1.15 (1.01-1.30)	
Physical activity		0.934		0.899
Light	1.13 (0.92-1.38)		1.14 (0.90-1.43)	
Moderate to vigorous	1.17 (1.02-1.36)		1.14 (0.99-1.32)	
BMI		0.744		0.851
<30	1.10 (0.91-1.34)		1.13 (0.94-1.37)	
≥30	1.17 (1.01-1.35)		1.13 (0.99-1.30)	
Diabetes		0.051		0.155
No	1.22 (1.08-1.38)		1.18 (1.02-1.36)	
Yes	0.97 (0.74-1.27)		1.02 (0.74-1.43)	
HBV and HCV				
HBV negative	1.16 (1.03-1.31)		1.15 (1.00-1.31)	
HCV negative	1.16 (1.02-1.31)		1.14 (1.00-1.31)	
Both negative	1.17 (1.03-1.33)		1.16 (1.01-1.32)	

Fig. 3 Subgroup analysis of associations between per 1-SD increase of intake of total starchy vegetables and potatoes and odds of hepatic steatosis (CAP ≥ 290 dB/m) in NHANES (2017–2018). *BMI* body mass index, *CAP* controlled attenuation parameter, *CI* confidence interval, *HBV* hepatitis B virus, *HCV* hepatitis C virus, *METS* metabolic equivalent tasks, *NHANES* National Health and Nutrition Examination Survey, *OR* odds ratio. Covariates adjusted in the models were the same as those in model 2 in Table 2 (see footnote in Table 2). Of note, the variables examined in this figure were not adjusted. Light physical activity was defined as participants with physical activity less than 8.3 METS-h per week, and moderate to vigorous activity was defined as participants who had a physical activity of 8.3 METS-h per week or more. We excluded participants with any missing values in covariates

associated with higher prevalence of both hepatic steatosis and fibrosis [10].

Second, oxidative stress is an important mediator in CLD pathogenesis and progression. Green leafy vegetables can reduce oxidative stress because they contain several antioxidant nutrients, minerals, phytochemicals, flavonoids, phenols, or dietary fiber [18, 20]. For example, an animal study showed that dietary nitrate, which was found in high levels in green leafy vegetables, could attenuate the progression of hepatic steatosis [24]. Although potatoes are also rich in antioxidant ingredients, such as ascorbic and chlorogenic acids [25], these antioxidants would be significantly reduced in cooking treatments and peeling [26]. Likewise, the antioxidant activity of other starchy vegetables, such as peas, may be markedly decreased during the canning process [4]. Additionally, long-term intake of acrylamide-containing potato chips may induce oxidative stress in humans by activating leukocytes and increasing the generation of reactive oxygen radicals [27].

Third, chronic inflammation has been implicated in the pathogenesis of CLD. Although intake of pigmented potatoes, such as yellow and purple-flesh potatoes, can reduce inflammation, white potato consumption may increase the plasma CRP concentration [28]. A study of 1158 participants used reduced rank regression with CRP and Interleukin 6 (IL-6) as response variables to derive a pro-inflammatory dietary pattern, and found that potato was one of the pro-inflammatory foods [29]. In addition, intake of potato chips may increase IL-6 and CRP concentrations, possibly because acrylamide contained in potato chips can lower the reserves of glutathione, an important cellular antioxidant [27]. However, antioxidants contained in non-starchy vegetables, such as polyphenols and dietary nitrate, have anti-inflammatory effects [7, 18]. Our results also suggested that intake of starchy vegetables was associated with higher hs-CRP level compared with non-starchy vegetable.

Fourth, obesity is an independent risk factor for CLD. Previous studies found that high potato intake [30] was positively, whereas increased intake of non-starchy vegetables [1] was inversely associated with waist circumference and weight gain. As mentioned above, starchy vegetables have higher energy content from starch and higher GL than non-starchy vegetables [1, 20]. Therefore, the positive association between starchy vegetables and weight-related outcomes may be caused by energy and fat deposition promoted by the higher insulin response. Conversely, non-starchy vegetables have higher water and fiber content, which can promote satiety and assist in weight management. Moreover, increased intake of green leafy vegetables which are rich in nitrate conveys an anti-obesity effect. Therefore, weight gain associated with starchy vegetable intake may also be a potential mechanism for the development of hepatic steatosis and fibrosis.

Strengths of our study include a large nationally representative sample of US adults and a valid noninvasive TE detection to measure hepatic steatosis and fibrosis [12]. However, several limitations should be noted. First, measurement error was unavoidable for self-reported diet and other lifestyle factors. In addition, two 24-h recalls may not well capture long-term dietary habits, although the NHANES design used several methods, such as the dietary sampling weight and multiple-pass method, to reduce measurement error and improve estimate of dietary intake. Second, we were unable to consider cooking methods in the analysis, which may lead to confounding bias, because previous studies suggested that different cooking/processing methods might influence nutrient content and GI value of potatoes [25]. Third, cross-sectional design in the current study cannot establish causal relationship. Fourth, despite the nationally representative sample in the current study, the generalizability of our results to other populations, such as Asia–Pacific populations, is limited, given the differences in food composition and cooking or processing method across regions or countries.

In summary, our results suggest that total starchy vegetable and potato intake are associated with higher, whereas total non-starchy and dark-green vegetable intake are associated with lower odds of steatosis and/or fibrosis, and provide further food-specific guidance for preventing CLD. Given the cross-sectional design in the current study, cohort studies or clinical trials from diverse populations that carefully consider cooking/processing methods of vegetables are warranted to validate our findings.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s12072-022-10368-x.

Acknowledgements This work was supported by the National Natural Science Foundation of China (82073651), Anhui Provincial Natural Science Foundation (2008085MH262 and 2108085QH357), Anhui Provincial Education Department (gxyqZD2021099), and grants from Anhui Medical University (XJ201935, 2020lcxk033, and 2021xkjT007). The funding agency had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Author contributions XL, TZ, and HL had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: WY and XL. Acquisition, analysis, or interpretation of data: all authors. Drafting of the manuscript: XL and WY. Critical revision of the manuscript for important intellectual content: all authors. Statistical analysis: XL, TZ, and HL. Obtained funding: WY. Administrative, technical, or material support: WY. Study supervision: WY.

Funding This work was supported by the National Natural Science Foundation of China (82073651), Anhui Provincial Natural Science Foundation (2008085MH262 and 2108085QH357), Anhui Provincial Education Department (gxyqZD2021099), and grants from Anhui Medical University (XJ201935, 2020lcxk033, and 2021xkjT007).

Availability of data and materials The data of this study were from public data; it will be made available to other researchers. The data were made available at https://wwwn.cdc.gov/nchs/nhanes/continuousnhanes/default.aspx?BeginYear=2017.

Declarations

Conflict of interest Xiude Li, Tengfei Zhang, Haowei Li, Zhihao Zhou, Meiling Li, Xueke Zeng, Hu Yang, Mingyi Zhang, Yong Huang, Yu Zhu, Zhuang Zhang, Yanan Ma and Wanshui Yang declare that they have no conflict of interest.

Ethics approval The NCHS Research Ethics Review Board approved the NHANES study protocols (Protocol #2011–17; Protocol #2018–01).

Animal research This was not animal research.

Consent to participate The written informed consent was obtained from all the participants.

Consent for publication All the authors consented the publish work.

References

1. Bertoia ML, Mukamal KJ, Cahill LE, et al. Changes in intake of fruits and vegetables and weight change in United States men and women followed for up to 24 years: analysis from three prospective cohort studies. PLoS Med. 2015;12(9): e1001878
2. Wang DD, Li Y, Bhupathiraju SN, et al. Fruit and vegetable intake and mortality: results from 2 prospective cohort studies of US men and women and a meta-analysis of 26 cohort studies. Circulation. 2021;143(17):1642–1654
3. Barclay AW, Petocz P, McMillan-Price J, et al. Glycemic index, glycemic load, and chronic disease risk–a meta-analysis of observational studies. Am J Clin Nutr. 2008;87(3):627–637
4. Murcia MA, Jimenez AM, Martinez-Tome M. Vegetables antioxidant losses during industrial processing and refrigerated storage. Food Res Int. 2009;42(8):1046–1052
5. Tajima R, Kimura T, Enomoto A, et al. No association between fruits or vegetables and non-alcoholic fatty liver disease in middle-aged men and women. Nutrition. 2019;61:119–124
6. Shi L, Liu ZW, Li Y, et al. The prevalence of nonalcoholic fatty liver disease and its association with lifestyle/dietary habits among university faculty and staff in Chengdu. Biomed Environ Sci. 2012;25(4):383–391

7. Kim SA, Shin S. Fruit and vegetable consumption and non-alcoholic fatty liver disease among Korean adults: a prospective cohort study. J Epidemiol Community Health. 2020;74(12):1035–1042
8. US Department of Agriculture and US Department of Health and Human Services. Dietary guidelines for Americans (2020–2025). 2021; https://www.dietaryguidelinesgov/resources/2020-2025-dietary-guidelines-online-materials. Accessed 18 Dec 2021
9. Health Canada. Canada's Dietary Guidelines. 2020; https://food-guide.canada.ca/en/guidelines/appendix-b-summary-guidelines-and-considerations/. Accessed 5 Jan 2022
10. Zhu Y, Peng Z, Lu Y, et al. Higher dietary insulinaemic potential is associated with increased risk of liver steatosis and fibrosis. Liver Int. 2022;42(1):69–79
11. Centers for Disease Control and Prevention. Glossary—Alcohol. https://www.cdc.gov/nchs/nhis/alcohol/alcohol_glossary.htm. Accessed 19 Apr 2022
12. Eddowes PJ, Sasso M, Allison M, et al. Accuracy of FibroScan controlled attenuation parameter and liver stiffness measurement in assessing steatosis and fibrosis in patients with nonalcoholic fatty liver disease. Gastroenterology. 2019;156(6):1717–1730
13. Song M, Giovannucci E. Substitution analysis in nutritional epidemiology: proceed with caution. Eur J Epidemiol. 2018;33(2):137–140
14. Caussy C, Alquiraish MH, Nguyen P, et al. Optimal threshold of controlled attenuation parameter with MRI-PDFF as the gold standard for the detection of hepatic steatosis. Hepatology. 2018;67(4):1348–1359
15. Petta S, Wong VW, Camma C, et al. Improved noninvasive prediction of liver fibrosis by liver stiffness measurement in patients with nonalcoholic fatty liver disease accounting for controlled attenuation parameter values. Hepatology. 2017;65(4):1145–1155
16. Li C, Guo P, Okekunle AP, et al. Lean non-alcoholic fatty liver disease patients had comparable total caloric, carbohydrate, protein, fat, iron, sleep duration and overtime work as obese non-alcoholic fatty liver disease patients. J Gastroenterol Hepatol. 2019;34(1):256–262
17. Li H, Wang X, Ye M, et al. Does a high intake of green leafy vegetables protect from NAFLD? Evidence from a large population study. Nutr Metab Cardiovasc Dis. 2021;31(6):1691–1701
18. Mokhtari E, Farhadnejad H, Salehi-Sahlabadi A, et al. Spinach consumption and nonalcoholic fatty liver disease among adults: a case-control study. BMC Gastroenterol. 2021;21(1):196
19. Cook LT, O'Reilly GA, Goran MI, Weigensberg MJ, Spruijt-Metz D, Davis JN. Vegetable consumption is linked to decreased visceral and liver fat and improved insulin resistance in overweight Latino youth. J Acad Nutr Diet. 2014;114(11):1776–1783
20. Yu EY, Wesselius A, Mehrkanoon S, et al. Vegetable intake and the risk of bladder cancer in the BLadder Cancer Epidemiology and Nutritional Determinants (BLEND) international study. BMC Med. 2021;19(1):56
21. Valtuena S, Pellegrini N, Ardigo D, et al. Dietary glycemic index and liver steatosis. Am J Clin Nutr. 2006;84(1):136–142 (**quiz 268-139**)
22. Bawden S, Stephenson M, Falcone Y, et al. Increased liver fat and glycogen stores after consumption of high versus low glycaemic index food: a randomized crossover study. Diabetes Obes Metab. 2017;19(1):70–77
23. Bertani JPB, Alves BC, Azevedo LA, Alvares-da-Silva MR, Dall'Alba V. Is dietary glycemic load associated with liver fibrosis in hepatitis C? Nutr Hosp. 2018;35(1):140–147
24. Liu Y, Croft KD, Caparros-Martin J, O'Gara F, Mori TA, Ward NC. Beneficial effects of inorganic nitrate in non-alcoholic fatty liver disease. Arch Biochem Biophys. 2021;711: 109032
25. Camire ME, Kubow S, Donnelly DJ. Potatoes and human health. Crit Rev Food Sci Nutr. 2009;49(10):823–840
26. Akyol H, Riciputi Y, Capanoglu E, Caboni MF, Verardo V. Phenolic compounds in the potato and Its byproducts: an overview. Int J Mol Sci. 2016;17(6):835
27. Naruszewicz M, Zapolska-Downar D, Kosmider A, et al. Chronic intake of potato chips in humans increases the production of reactive oxygen radicals by leukocytes and increases plasma C-reactive protein: a pilot study. Am J Clin Nutr. 2009;89(3):773–777
28. Kaspar KL, Park JS, Brown CR, Mathison BD, Navarre DA, Chew BP. Pigmented potato consumption alters oxidative stress and inflammatory damage in men. J Nutr. 2011;141(1):108–111
29. Barbaresko J, Rienks J, Oluwagbemigun K, et al. Dietary patterns associated with inflammatory biomarkers in a Northern German population. Eur J Nutr. 2020;59(4):1433–1441
30. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. N Engl J Med. 2011;364(25):2392–2404

Authors and Affiliations

Xiude Li[1,2,3,4] · Tengfei Zhang[1] · Haowei Li[1] · Zhihao Zhou[1] · Meiling Li[1] · Xueke Zeng[1] · Hu Yang[1] · Mingyi Zhang[1] · Yong Huang[1] · Yu Zhu[1] · Zhuang Zhang[1] · Yanan Ma[5] · Wanshui Yang[1,2,3,4]

Xiude Li
xiudeli@foxmail.com

Tengfei Zhang
792764793@qq.com

Haowei Li
lhw0901@foxmail.com

Zhihao Zhou
2298181959@qq.com

Meiling Li
limeiling3423@163.com

Xueke Zeng
nicolezxk@163.com

Hu Yang
huyang5717@163.com

Mingyi Zhang
1183133820@qq.com

Yong Huang
topgun_hy@aliyun.com

Yu Zhu
kutuomonk@foxmail.com

Zhuang Zhang
zhzh_ahmu@outlook.com

[1] Department of Nutrition, School of Public Health, Anhui Medical University, 81 Meishan Road, Hefei 230032, Anhui, China

[2] Key Laboratory of Population Health Across Life Cycle (Anhui Medical University), Ministry of Education of the People's Republic of China, Hefei, Anhui, China

[3] NHC Key Laboratory of Study on Abnormal Gametes and Reproductive Tract, Hefei, Anhui, China

[4] Anhui Provincial Key Laboratory of Population Health and Aristogenics/Key Laboratory of Environmental Toxicology of Anhui Higher Education Institutes, Anhui Medical University, Hefei, Anhui, China

[5] Department of Biostatistics and Epidemiology, School of Public Health, China Medical University, No. 77 Puhe Road, Shenyang North New Area, Shenyang 110122, Liaoning Province, China