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Coffee consumption is associated with lower liver stiffness: a nationally representative study

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Abstract

Background: Coffee is associated with a reduced risk of liver disease. This association is limited by important sources of confounding such as recall bias, healthy-user bias, and indirect measures of liver outcomes or health. We aimed to examine the impact of coffee consumption with liver fibrosis and steatosis in a nationally representative sample.

Methods: We evaluated 4,510 subjects ≥20 years old from the 2017–2018 NHANES study that underwent both transient elastography and two 24-hour dietary recall examinations. We tested the associations between liver stiffness measurements (LSM) ≥ 9.5 kpa or controlled attenuation parameter (CAP) and coffee consumption. We used decaffeinated coffee and tea consumption as controls. As sensitivity analysis, we included all drinks in one model, examined the impact of caffeine consumption, and adjusted for the Healthy Eating Index-2015 (HEI-2015) and sugar-sweetened beverage consumption as separate models.

Results: The study sample described was aged 48 ± 0.6 years, 73% were overweight or obese, 10.6% had diabetes, 47.5% reported participation in vigorous physical activity, and 23% drank ≥ 2 alcoholic drinks per day. After multivariate adjustment, there was no association between coffee and controls with CAP. Subjects who drank >3 cups of coffee, but not other drinks, had 0.9 lower kPa (95% CI −1.6 – −0.1, p = 0.03). >3 cups of coffee were protective for LSM ≥

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1. is the guarantor of this article
2. Roles
a. Concept: Tapper
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e. Revision: Jiang

9.5 kpa (OR: 0.4, 95% CI 0.2 – 1.0, p = 0.05). Accounting for all beverages in the same model, only >3 cups of coffee remained independently associated with LSM (OR: 0.5, 95% CI 0.2 – 0.9, p= 0–03). Caffeine was not significantly associated with LSM at any dose. Finally, adjusting for sugar-sweetened beverage consumption and HEI-2015, coffee consumption remained associated with lower LSM. The protective nature of coffee consumption is therefore not attributable to caffeine and persists in participants regardless of their diet quality.

Conclusion: Coffee is associated with lower liver stiffness but not steatosis as measured by CAP among US adults.

Keywords

NHANES; fatty liver; caffeine; liver disease

Introduction

Therapies to prevent or forestall the progression of cirrhosis toward decompensation are lacking. Ideally, we would treat the underlying liver disease to prevent the development of cirrhosis. However, once a patient's liver becomes cirrhotic, they remain at high risk of decompensation. The lifetime risk of ascites is 50% and hepatic encephalopathy is 40%.[1] There is, accordingly, great enthusiasm for therapies that are associated with reduced risk of incident cirrhosis. In particular, there is substantial observational data suggesting that coffee consumption is associated with a lower risk of cirrhosis.[2–6]

The biases of observational data are real and must be addressed. In studies of nutritional exposures, matching treated to untreated patients introduces 3 major limits on the value of observational data. First, healthy-user bias[7]: subjects more likely to exhibit healthier behavior may consume more coffee or those who are presently not consuming coffee any longer are no longer healthy. Second, recall bias: many studies of coffee consumption use non-ideal methods to ascertain exposure history. Third, selection bias: persons undergoing evaluation for liver disease may be fundamentally different from those who are not. Accordingly, studies that match cases to controls or use biopsy findings cannot be representative of the general population.

Herein, we evaluate a large, nationally representative cohort of Americans who underwent both vibration-controlled transient elastography as well as the gold standard for nutritional epidemiology, two 24-hour dietary recalls. We previously showed that sugar sweetened beverages were associated with higher liver stiffness and steatosis measurements.[8] In this study, we examine the effect of coffee consumption on liver stiffness accounting for diet quality, sugary beverages, and comparing it to the effect of consumption of caffeine, tea, or decaffeinated coffee.

Methods

Study population

NHANES is a nationally representative cross-sectional study that enrolls participants through a stratified multistage probability and oversampling design that allows weighted

analysis that represents the civilian non-institutionalized US population. The participants are interviewed for demographic, socioeconomics, health, and dietary information using a general questionnaire format and two twenty-four-hour recalls conducted as a partnership between the US Department of Agriculture (USDA) and the US Department of Health and Human Services (DHHS). The first dietary recall is administered in person at the NHANES Mobile Examination Center and the second dietary recall is administered over the phone 3–10 days later. All interviewers are required to complete an intensive one-week training course and conduct supervised practice interviews before doing these independently for the recalls. Quality control is employed for completeness of recalls, missing information, inconsistent reports, and unclear notes. No proxy responses were allowed. A set of measuring guides including measuring cups, spoons, glasses, and bottles, were used as visual aids for the participant to use for reporting amounts of food or drinks consumed. The 24-hour dietary recall collects information on all foods and beverages consumed during the previous day using the USDA Automated Multiple Pass Method. Examinations and laboratory tests are conducted on a subset of participants. We excluded all subjects with viral hepatitis. The cohort construction flowchart is shown in Supplementary Figure 1.

Measurement of liver stiffness and liver steatosis

We included 4,510 participants that were aged 20 years or older that had a complete elastography exam. According to NHANES, an exam was considered complete with 10 or more complete stiffness (E) measurements, fasting time of at least three hours, and if the liver stiffness interquartile (IQRe) range/median E was less than 30%. Liver stiffness measurement (LSM) was dichotomized using 9.5 kPa as a threshold for advanced fibrosis according to the literature.[9] Controlled attenuation parameter (CAP) was considered as a continuous variable.

Background information and dietary recall data

We obtained self-reported information on age, gender, ethnicity, alcohol consumption in the past 12 months, diabetes history, smoking history, education level, and vigorous physical activity. Alcohol consumption was categorized into 6 groups based on daily consumption: never drinkers or no drinks in the last year, former heavy drinkers, <1 drink, 1–2 drinks, 2–4 drinks, and 5+ drinks. Body mass index (BMI) was calculated using the body weight and height measurements from the examination portion of the survey.

Dietary intake data was extracted from the two 24 dietary-recall interviews. Coffee, decaffeinated coffee, and tea intake were transformed into 6-ounce servings and categorized based on number of cups consumed (non-drinkers, <1 cup, 1–2 cups, 2–3 cups, >3 cups). Caffeine consumption from any source reported by participants was transformed into milligrams (mg) using the USDA's Food and Nutrition Database for Dietary Studies (FNDDS). We categorized caffeine consumption into 5 groups: non-drinkers, <100 mg, 100–200 mg, 200–300 mg, and >300 mg. Sugar sweetened beverages (SSBs) included soft drinks, fruit drinks with added sugar, sweetened coffee and tea drinks, sport drinks, and sweetened bottled water. Intake of 100% fruit juice, unsweetened milk, flavored milk, coffee, and tea were not categorized as SSBs. We also calculated the Healthy Eating Index (HEI)-2015 score, a measure of overall diet quality.[10]

Statistical analysis

For the main analysis, multivariable linear regression was performed to determine the associations between consumption of coffee (Model 1), decaffeinated coffee (Model 2), tea (Model 3) and CAP (dB/m). Only linear regression was presented for CAP because there was no overall association. Given an association with coffee and LSM, we present both the multivariable linear regression and the logistic regression with liver stiffness (LSM≥9.5 kPa), for ease of clinical interpretation. Each model was adjusted for age (every 10 years), gender, race/ethnicity, education level, vigorous physical activity, BMI (every 5 points), smoking history, diabetes, and alcohol consumption in the past 12 months. We performed 4 sensitivity tests. First, we evaluated associations with caffeine consumption (grams). Second, we evaluated associations accounting for sugar sweetened beverage consumption as an explicit marker of diet quality given its known association with LSM.[8] The associations between HEI and LSM were also evaluated. Third, we combined coffee, decaffeinated coffee, and tea in one model to determine independent associations with CAP and LSM. All data analyses were conducted using R and Stata version 16.1 with NHANES-provided sampling weights

Results

General description

The study sample is described in Table 1. Participants were aged 48 ± 0.6 years, half were women, 1 in 3 were college educated, and 3 in 5 were white. Three in four were overweight or obese, 1 in 10 had diabetes, half reported participation in vigorous physical activity, and 3 in 4 reported any alcohol consumptions. Overall, 3,797 subjects had LSM<7.0kPa, 415 LSM7–9.5kPa, and 298 LSM≥9.5kpa. In Supplementary Table 1 we compare cohort characteristics according to coffee consumption. Coffee consumption does not appear to be associated with other health indicators and is associated with higher rates of obesity, diabetes, and alcohol consumption.

Associations with CAP

We first assessed the relationship between consumption of coffee, decaffeinated coffee, and tea on CAP (Table 2). There was no association between any drink and CAP. For each model, several factors were consistently associated with increased CAP: age, Hispanic and Asian race, BMI, diabetes, high school education. Conversely, Black race, female sex, and vigorous physical actively were associated with lower CAP.

Associations with LSM

We then examined the relationship between consumption of coffee, decaffeinated coffee, and tea on LSM (Table 3). There was a significant association between subjects who drank >3 cups of coffee and decreased LSM, with a beta-coefficient of −0.9 (95% CI −1.6 — −0.1, p=0.03). In contrast, there was no significant association between LSM and both decaffeinated coffee and tea. For each model, several factors were consistently associated with increased LSM: BMI, diabetes, alcohol consumption (J-shaped association) and lack

thereof. Conversely, Black race and female sex were consistently associated with decreased LSM.

In modelling that focused on LSM≥9.5kPa (Supplementary Table 2), subjects who drank >3 cups of either coffee or tea had lower risk of LSM≥9.5, with respective odds ratios of 0.4 (95% CI 0.2–1.0, p=0.05). For each model, several factors were associated with increased odds of LSM≥9.5. These included age, BMI, diabetes, and alcohol consumption (2–4 drinks/day). Conversely, females had lower odds of LSM≥9.5.

Sensitivity testing

First, the similarity of significant association of coffee and tea consumption on LSM prompted an additional analysis where coffee, decaffeinated coffee, and tea were covariates in the same model (Table 4). In this logistic regression analysis, only >3 cups of coffee remained significantly associated with LSM (OR 0.5, 95%CI 0.2–0.9, p=0.03). All other stratified drink categories, including >3 cups of tea, were not significantly associated with LSM.

Second, since both coffee and tea were significantly associated with the LSM in individual modelling - but decaffeinated coffee was not - caffeine's relationship to LSM was investigated (Supplementary Table 3). In this logistic regression sensitivity analysis, caffeine was not significantly associated with LSM for all stratified amounts: no caffeine, <100 mg, 100–200 mg, 200–300 mg, and >300 mg.

Third, to account for other markers of diet quality, we evaluated associations when adjusting for the Healthy Eating Index-2015 and sugar-sweetened drink consumption (Supplementary Tables 4–5). Accounting for the Healthy Eating Index-2015, >3 cups of coffee daily was associated with lower LSM – beta −1.0 (95% CI-1.8 – −0.1, p=0.02) – with a trend for LSM≥9.5 kPa – OR 0.4 (95% CI 0.2–1.1, p=0.06). Coffee consumption remained associated with LSM≥9.5 when adjusting for sugar-sweetened drink consumption, with increased statistical significance: >3 cups had an OR of 0.4 (95% CI 0.1–0.9, p=0.03).

Discussion

Coffee is reproducibly associated with improved liver-related outcomes among persons with or at risk for liver disease. These associations, however, are confounded by biases inextricably linked to observation study designs. In this nationally representative cross-sectional study of American adults, we clarify association between coffee and liver disease in several ways. First, we show that coffee consumption is associated with liver stiffness but not liver steatosis. Second, our data is uniquely free of selection bias for the outcome of interest (elastography) and our subjects are both diverse and nationally representative. Third, we use two 24-hour dietary recalls, the gold-standard in nutritional epidemiology, to quantify coffee consumption overcoming recall bias. Finally, we use a variety of methods to address potential confounding including control comparisons (decaffeinated coffee, tea) and robust adjustment for lifestyle (physical activity, sugar-sweetened beverage consumption, and overall dietary quality).

Coffee is associated with lower liver stiffness

In our study, we find that >3 cups of coffee daily were associated with reduced liver stiffness accounting for lifestyle confounders. People who drink >3 cups experience reduced risk associated with elevated liver stiffness ≥9.5 kPa, OR 0.4 (95% CI 0.2–1.0). Adjusting for consumption of decaffeinated coffee and tea, this effect is unchanged, OR 0.5 (95% CI 0.2–0.9). We previously showed that sugar-sweetened beverages are associated with elevated liver stiffness and CAP.[8] When adjusting for sugary-drink consumption, the effect of coffee is also unchanged, OR 0.4 95%CI(0.1–0.9). The overall association persists when adjusting for the Healthy Eating Index but was slightly attenuated for the LSM≥9.5 kPa cutoff. This effect was not seen with decaffeinated coffee. However, although tea was not associated with LSM in the linear regression, among subjects who consumed >3 cups of tea there was a signal, OR 0.4 (95% CI 0.4–1.0). We therefore assessed the association between caffeine and liver stiffness. Though we find that caffeine was not significantly associated with liver stiffness, the expected caffeine concentration in coffee required for analysis of 24-hour dietary inventories may vary based on its preparation. Given the discordant associations between caffeinated and de-caffeinated coffee, if these associations are true, caffeine's role in the mechanism cannot be excluded on the basis of these data.

Coffee as antifibrotic candidate

We find that coffee is associated with a lower risk of elevated liver stiffness but not fatty liver. These findings echo the strongest prior data. Coffee is associated with a lower risk of cirrhosis and its complications, including hepatocellular carcinoma.[2–6,11,12] If not attributable to confounding, the mechanisms underlying these associations are unclear. Though some have suggested that coffee is associated with a reduced risk of fatty liver disease and, broadly, hepatic steatosis,[13] our study is not supportive. We find no association between coffee, caffeinated or decaffeinated, and CAP, a highly sensitive measure of liver fat. Instead, the evidence for coffee as an antifibrotic is stronger. First, caffeine is an adenosine receptor antagonist, which reduces adenosinergic fibrogenesis.[14,15] Second, in a small 40-person 1:1 randomized trial of 4 cups of coffee versus none among subjects with chronic hepatitis C, coffee was associated with reduced collagen production.[16]

Study Strengths

Our study extends this field by evaluating the cross-sectional association between liver stiffness and coffee-consumption with unique strengths. First, whereas prior population-based studies have used cursory coffee consumption surveys or food frequency questionnaires,[3,12,16] our data is derived from two 24-hour dietary recalls, the most accurate and complete description of dietary intake.[16] Second, our study sample is nationally representative. Many prior studies have recruited biased samples of patients undergoing liver biopsy or participating in clinical trials.[3,5] Third, we used transient-elastography to quantify liver health at the time of the 24-hour dietary recalls. While population-based studies have been conducted using food frequency questionnaires, they have relied on inaccurate measures to categorize liver disease such as diagnostic codes[12] or indirect estimations of liver disease such as liver enzymes,[17] transient elastography is a direct assessment of liver characteristics.

Contextual factors

These data must be interpreted in the context of the study design. Fist, our data are cross-sectional and neither causality nor associations with clinical outcomes can be inferred. Second, those with failed VCTE exams (including high IQR/m) were not captured in the study and could represent a significant at-risk group. Third, unmeasured confounding is possible that could acc. For lack of a clear mechanism Finally, all studies of diet are at risk of bias. While 24-hour recalls by trained interviewers enrich the quality of the data and limit the risk of recall bias, the main pitfall is that the 2 days evaluated may not be representative of the subject's overall diet behaviour.

Conclusions

Coffee is associated with lower liver stiffness. In the absence of randomized trials, these cross-sectional, nationally representative data employing direct measures of liver health and gold standard dietary inventories provide some of the strongest possible evidence for this association.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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"What you need to know"

BACKGROUND

Coffee is associated with a reduced risk of advanced liver disease and its complications. However most studies are confounded by selection bias and employ indirect measures of liver function and outcomes.

FINDINGS

In this study a nationally representative cohort of American subjects who underwent 24-hour dietary recalls and transient elastrography as part of NHANES, we find that >3 cups of coffee daily are associated with lower liver stiffness.

IMPLICATIONS FOR PATIENT CARE

These data strengthen the association between coffee consumption and a lower risk of liver disease is strengthened.

Table 1.

Population estimates and observations of eligible population

Characteristic		Pop. Estimates	Observations
Age – years (SD)		47.9 (0.6)	
Gender	Male	102,527,879.40 (49.3%)	2,233
	Female	105,272,850.60 (50.7%)	2,277
Race	White	129,828,692.50 (62.5%)	1,527
	Black	23,267,656.40 (11.2 %)	1,049
	Hispanic	32,851,134.50 (15.8%)	1,038
	Asian	12,071,417.30 (5.8%)	655
	Other	9,781,829.40 (4.7%)	241
Body Mass Index	Underweight (< 18.5)	3,032,192.60 (1.5%)	67
	Normal weight (18.5 – 24.9)	53,319,343.70 (25.8%)	1,129
	Overweight (25.0 – 29.9)	65,001,041.40 (31.5%)	1,467
	Obese (> 30.0)	85,253,361.40 (41.3%)	1,809
Diabetes		21,962,459.80 (10.6%)	679
Hypercholesterolemia		68,270,536.00 (33.0%)	1,594
Heart Failure		3,359,226.8 (1.6%)	111
Hypertension		87,137,938.5 (43.4%)	2,222
Coronary Heart Disease		7,204,802.8 (3.5%)	181
Angina		4,620,892.5 (2.2%)	113
Myocardial Infarction		6,172,784.5 (3.0%)	188
Stroke		5,657,364.8 (2.7%)	192
Chronic Obstructive Pulmonary Disease		7,864,503.8 (3.8%)	202
Thyroid Condition		19,248,324.5 (9.3%)	388
Chronic Liver Disease		10,162,840.4 (4.9%)	246
Education	Less than high school	22,876,540.70 (11.0%)	880
	High school graduate	55,728,703.20 (26.8%)	1,061
	Some college or AA degree	63,723,778.10 (30.6%)	1,453
	College or above	65,324,836.60 (31.5%)	1,108
Vigorous Physical Activity		98,567,215.20 (47.5%)	1,857
Daily Alcohol Drinks in last 12 months	Never/none in last year	48,443,997.80 (23.4%)	1,388
	Former heavy drinker	7,340,091.10 (3.5%)	176
	1 drink or less	53,833,725.20 (26.0%)	1,098
	1 to 2 drinks	48,116,958.80 (23.2%)	896
	2 to 4 drinks	32,593,063.30 (15.7%)	605
	5 drinks or more	17,103,390.80 (8.3%)	342
Daily Cups of Coffee	No cups	46,753,573.2 (25.4%)	1,072
	<1 cup	10,134,545.9 (5.5%)	244
	1–2 cups	54,891,552.6 (3.0%)	1,267

Characteristic		Pop. Estimates	Observations
	2–3 cups	41,286,723.7 (22.4%)	785
	>3	31,369,199.9 (17.0%)	515
Daily Cups of Decaffeinated Coffee	No cups	183,076,890.7 (93.6%)	3,883
	<1 cup	1,019,019.1 (0.5%)	33
	1–2 cups	5,397,806.7 (2.8%)	127
	2–3 cups	3,417,207.9 (1.8%)	69
	>3	2,614,299.6 (1.3%)	38
Daily Cups of Tea	No cups	118,733,107.8 (60.7%)	2,593
	<1 cup	5,876,738.9 (3.0%)	3,430
	1–2 cups	28,139,327.0 (14.4%)	372
	2–3 cups	23,259,073.5 (11.9%)	206
	>3	19,516,976.8 (10.0%)	142
Daily Cups of Sugar Sweetened Beverages (SSBs)	No cups	102,684,035.8 (58.9%)	2,144
	<1 cup	5,847,400.0 (3.3%)	147
	1–2 cups	28,899,095.7 (16.3%)	688
	2–3 cups	23,183,212.4 (13.1%)	439
	>3	16,764,410.4 (9.5%)	300
Healthy Eating Index (HEI)		53.4 (0.7)	3,634
Caffeine (mg)		168.2 (5.0)	3,634

*Data are presented as mean (SD) for continuous measures, and n (%) for categorical measures.

Table 2.

Adjusted associations with Controlled Attenuation Parameter (dB/m)

Characteristic		Coffee Model 1[2] (Coef. (95% CI), p)		Decaffeinated Coffee Model 2[2] (Coef. (95% CI), p)		Tea Model 3[2] (Coef. (95% CI), p)	
Age (Every 10 years)		5.9 (4.6 – 7.3)	<0.001	5.9 (4.6 – 7.1)	<0.001	5.8 (4.6 – 7.1)	<0.001
Female (Ref. = Male)		−19.9 (−24.3 – −15.4)	<0.001	−20.8 (−25.1 – −16.5)	<0.001	−20.9 (−25.2 – −16.7)	<0.001
Race (Ref. = White)	Black	−21.9 (−28.9 – −14.9)	<0.001	−21.0 (−26.5 – −15.6)	<0.001	−21.0 (−26.4 – −15.6)	<0.001
	Hispanic	8.0 (1.8 – 14.1)	0.01	7.5 (2.3 – 12.6)	0.007	7.6 (2.6 – 12.5)	0.006
	Asian	16.1 (10.2 – 22.1)	<0.001	15.8 (10.1 – 21.6)	<0.001	15.4 (8.7 – 22.2)	<0.001
	Others	−7.4 (−17.6 – 2.8)	0.1	−5.0 (−15.0 – 5.0)	0.3	−4.6 (−14.7 – 5.6)	0.3
Smoking (Ref. = Never)	Past smoker	2.3 (−3.0 – 7.5)	0.3	1.4 (−3.8 – 6.6)	0.5	1.5 (−3.6 – 6.6)	0.5
	Current smoker	3.6 (−1.5 – 8.8)	0.1	2.4 (−2.4 – 7.1)	0.3	2.7 (−1.7 – 7.1)	0.2
Vigorous activity		−8.9 (−12.9 – −4.9)	<0.001	−8.9 (−12.3 – −5.3)	0.001	−8.9 (−12.7 – −5.1)	0.001
BMI (kg/m^2) (every 5 pts.)		24.0 (21.6 – 26.4)	<0.001	24.1 (22.1 – 26.1)	<0.001	24.2 (22.2 – 26.2)	<0.001
Diabetes		20.1 (12.1 – 28.2)	<0.001	20.0 (12.2 – 27.6)	<0.001	20.0 (12.3 – 27.7)	<0.001
Coffee (Ref. = Non-drinkers)	<1 cup	0.2 (−9.0 – 9.5)	0.9	-		-	
	1–2 cups	−0.2 (−5.5 – 5.1)	0.9	-		-	
	2–3 cups	−2.8 (−8.8 – 3.2)	0.3	-		-	
	>3 cups	1.6 (−4.0 – 7.2)	0.5	-		-	
Decaffeinated coffee (Ref. = Non-drinkers)	<1 cup	-		−6.8 (−33.7 – 20.2)	0.6	-	
	1–2 cups	-		6.2 (−4.4 – 16.7)	0.2	-	
	2–3 cups	-		−3.8 (−18.4 – 10.8)	0.5	-	
	>3 cups	-		2.3 (−24.6 – 29.1)	0.8	-	
Tea (Ref. = Non-drinkers)	<1 cup	-		-		3.5 (−12.3 – 19.3)	0.6
	1–2 cups	-		-		2.7 (−3.7 – 9.1)	0.3
	2–3 cups	-		-		4.7 (−3.3 – 12.8)	0.2
	>3 cups	-		-		−1.2 (−10.5 – 8.2)	0.7

Characteristic		Coffee Model 1[2] (Coef. (95% CI), p)		Decaffeinated Coffee Model 2[2] (Coef. (95% CI), p)		Tea Model 3[2] (Coef. (95% CI), p)	
	High school	10.6 (6.7 – 14.4)	<0.001	9.2 (5.4 – 13.0)	0.001	9.1 (5.4 – 12.8)	<0.001
Education (Ref. = less than high school)	Some college	6.5 (0.7 – 12.4)	0.03	6.4 (0.8 – 12.0)	0.02	6.1 (0.4 – 11.9)	0.03
	College	4.4 (−2.2 – 10.9)	0.1	3.3 (−3.0 – 9.5)	0.2	3.0 (−3.3 – 9.3)	0.3
Number of alcohol drinks a day (Ref. = 1 drink or less)	Never/none in last year	1.7 (−6.7 – 10.1)	0.6	2.3 (−5.4 – 10.1)	0.5	2.4 (−5.7 – 10.5)	0.5
	Former heavy drinker	1.6 (−11.5 – 14.6)	0.8	1.3 (−12.4 – 15.1)	0.8	1.6 (−12.1 – 15.3)	0.8
	1 to 2	1.5 (−3.9 – 6.9)	0.5	1.8 (−3.4 – 7.0)	0.4	2.3 (−3.2 – 7.7)	0.3
	2 to 4	5.3 (−5.8 – 16.4)	0.3	6.7 (−3.4 – 16.8)	0.1	7.1 (−3.0 – 17.3)	0.1
	5 or more	8.4 (−3.9 – 20.7)	0.1	8.6 (−2.3 – 19.5)	0.1	9.1 (−1.8 – 20.0)	0.09

[1]Beta-coefficient calculated by linear regression for CAP dB/m.

[2]Adjusted model included age, gender, race, vigorous activity, alcohol consumption in the last year, smoking history, BMI, and education level.

Table 3.

Adjusted associations with liver stiffness measurements

Characteristic		Coffee Model 1[2] (β (95% CI), p)		Decaffeinated Coffee Model 2[2] β (95% CI), p)		Tea Model 3[2] (β (95% CI), p)	
Age (Every 10 years)		0.1 (−0.1 – 0.3)	0.1	0.1 (−0.1 – 0.3)	01	0.1 (−0.1 – 0.3)	0.1
Female (Ref. = Male)		−0.7 (−1.2 – −0.1)	0.03	−0.7 (−1.2 – −0.1)	0.01	−0.7 (−1.2 – −0.1)	0.01
Race (Ref. = White)	Black	−0.6 (−1.1 – −0.1)	0.03	−0.4 (−0.9 – 0.0)	0.07	−0.4 (−0.8 – 0.0)	0.05
	Hispanic	−0.5 (−1.0 – 0.1)	0.1	−0.4 (−1.0 – 0.2)	0.1	−0.4 (−0.9 – 0.2)	0.1
	Asian	0.1 (−0.3 – 0.4)	0.6	0.2 (−0.1 – 0.5)	0.2	0.2 (−0.1 – 0.6)	0.1
	Others	0.1 (−1.1 – 1.3)	0.8	0.1 (−0.8 – 1.0)	0.8	0.2 (−0.8 – 1.2)	0.7
Smoking (Ref. = Never)	Past smoker	0.1 (−0.4 – 0.5)	0.8	−0.0 (−0.5 – 0.5)	0.9	−0.0 (−0.5 – 0.4)	0.9
	Current smoker	0.2 (−0.4 – 2.5)	0.5	0.0 (−0.6 – 0.6)	0.9	0.1 (−0.4 – 0.6)	0.7
Vigorous activity		0.3 (−0.3 – 0.8)	0.3	0.2 (−0.4 – 0.8)	0.4	0.2 (−0.4 – 0.7)	0.5
BMI (kg/m^2) (every 5 pts.)		1.0 (0.7 – 1.3)	<0.001	1.0 (0.7 – 1.3)	<0.00 1	1.0 (0.7 – 1.3)	<0.001
Diabetes		1.4 (0.2 – 2.5)	0.02	1.4 (0.3 – 2.5)	0.01	1.4 (0.3 – 2.5)	0.01
Coffee (Ref. = Non-drinkers)	<1 cup	0.0 (−1.1 – 1.1)	0.9	-		-	
	1–2 cups	−0.3 (−0.9 – 0.2)	0.1	-		-	
	2–3 cups	−0.5 (−1.0 – 0.0)	0.06	-		-	
	>3 cups	−0.9 (−1.6 – −0.1)	0.03	-		-	
Decaffeinated coffee (Ref. = Non-drinkers)	<1 cup	-		0.0 (−1.2 – 1.2)	0.9	-	
	1–2 cups	-		0.4 (−0.4 – 1.3)	0.3	-	
	2–3 cups	-		−0.4 (−1.0 – 0.2)	0.1	-	
	>3 cups	-		1.1 (−2.3 – 4.6)	0.4	-	
Tea (Ref. = Non-drinkers)	<1 cup	-		-		−0.2 (−0.8 – 0.3)	0.3
	1–2 cups	-		-		−0.0 (−0.4 – 0.3)	0.8
	2–3 cups	-		-		0.7 (−0.6 – 1.9)	0.2
	>3 cups	-		-		−0.3 (−1.3 – 0.6)	0.4
Education (Ref. = less than high school)	High school	−0.4 (−1.7 – 0.9)	0.5	−0.4 (−1.5 – 0.8)	0.5	−0.4 (−1.6 – 0.8)	0.5
	Some college	−0.5 (−1.9 – 0.8)	0.4	−0.5 (−1.7 – 0.8)	0.4	−0.5 (−1.8 – 0.8)	0.4

Characteristic		Coffee Model 1[2] (β (95% CI), p)		Decaffeinated Coffee Model 2[2] β (95% CI), p)		Tea Model 3[2] (β (95% CI), p)	
	College	−0.9 (−2.1 – 0.4)	0.1	−0.8 (−2.0– 0.4)	0.1	0.9 (−2.1 – 0.4)	0.1
Number of alcohol drinks a day (Ref. = 1 drink or less)	Never/none in last year	0.4 (−0.0 – 0.8)	0.05	0.4 (0.1 – 0.8)	0.02	0.4 (0.0 – 0.8)	0.03
	Former heavy drinker	0.2 (−0.6 – 1.0)	0.6	0.1 (−0.7 – 0.9)	0.8	0.1 (−0.7 – 0.9)	0.7
	1 to 2	0.5 (0.2 – 0.9)	0.01	0.5 (0.0 – 0.9)	0.03	0.5 (0.1 – 0.9)	0.01
	2 to 4	0.9 (−0.1 – 1.9)	0.07	0.9 (−0.0 – 1.8)	0.05	1.0 (0.0 – 1.9)	0.04
	5 or more	0.2 (−0.9 – 1.4)	0.0	0.3 (−0.8 – 1.4)	0.5	0.3 (−0.8 – 1.4)	0.5

[1] Beta-coefficient calculated by linear regression for LSM (kPa)

[2] Adjusted model included age, gender, race, vigorous activity, alcohol consumption in the last year, smoking history, BMI, and education level.

Table 4.

Combined beverage models with liver stiffness measurements (LSM) ≥ 9.5 kPa and Controlled Attenuation Parameter (CAP, dB/m)

Characteristic		**All drinks and LSM≥9.5kPa Model 1[3] (OR (95% CI)[1], p)**		**All drinks and CAP (dB/m) Model 2[3] (Coef. (95% CI)[2], p)**	
Age (Every 10 years)		1.5 (1.3 – 1.7)	<0.001	6.1 (4.6 – 7.5)	<0.001
Female (Ref. = Male)		0.4 (0.3 – 0.8)	0.006	−20.0 (−24.4 – −15.6)	<0.001
Race (Ref. = White)	Black	0.8 (0.4 – 1.2)	0.2	−20.9 (−28.3 – −13.5)	<0.001
	Hispanic	1.1 (0.6 – 2.0)	0.7	8.5 (2.7 – 14.2)	0.007
	Asian	1.6 (0.7 – 3.6)	0.2	14.9 (7.9 – 21.8)	<0.001
	Others	0.9 (0.4 – 2.3)	0.8	−5.4 (−14.8 – 4.0)	0.2
Smoking (Ref. = Never)	Past smoker	0.8 (0.6 – 1.2)	0.2	2.5 (−2.6 – 7.7)	0.3
	Current smoker	1.6 (0.8 – 2.9)	0.1	4.4 (−0.4 – 9.1)	0.06
Vigorous activity		1.5 (0.9 – 2.6)	0.1	−8.8 (−13.1 – −4.6)	0.001
BMI (kg/m^2) (every 5 pts.)		2.2 (1.9 – 2.6)	<0.001	23.9 (21.4 – 26.4)	<0.001
Diabetes		2.6 (1.7 – 4.2)	<0.001	20.2 (12.2 – 28.3)	<0.001
Coffee (Ref. = Non-drinkers)	<1 cup	2.5 (0.8 – 7.5)	0.1	0.7 (−9.9 – 11.2)	0.8
	1–2 cups	1.0 (0.5 – 1.9)	0.9	−0.1 (−5.9 – 5.7)	0.9
	2–3 cups	1.1 (0.5 – 2.5)	0.7	−3.7 (−10.0 – 2.5)	0.2
	>3 cups	0.5 (0.2 – 0.9)	0.03	3.3 (−2.1 – 8.6)	0.2
Decaffeinated coffee (Ref. = Non-drinkers)	<1 cup	1.3 (0.3 – 6.0)	0.7	−8.5 (−33.9 – 17.0)	0.4
	1–2 cups	0.3 (0.1 – 1.3)	0.1	6.7 (−4.1 – 17.4)	0.2
	2–3 cups	0.8 (0.2 – 3.0)	0.7	−4.9 (−21.0 – 11.3)	0.5
	>3 cups	1.6 (0.5 – 5.3)	0.4	2.6 (−26.5 – 31.7)	0.8
Tea (Ref. = Non-drinkers)	<1 cup	0.5 (0.2 – 1.7)	0.2	5.3 (−11.6 – 22.1)	0.5
	1–2 cups	0.8 (0.5 – 1.4)	0.4	0.9 (−6.2 – 8.1)	0.7
	2–3 cups	1.2 (0.5 – 3.0)	0.6	7.9 (−1.1 – 17.0)	0.08
	>3 cups	0.6 (0.3 – 1.5)	0.2	−2.6 (−12.4 – 7.2)	0.6
Education (Ref. = less than high school)	High school	1.5 (0.8 – 2.8)	0.1	10.6 (6.1 – 15.1)	<0.001
	Some college	0.8 (0.4 – 1.4)	0.3	7.0 (0.6 – 13.4)	0.03
	College	0.5 (0.3 – 1.1)	0.08	4.6 (−1.7 – 11.0)	0.1
	Never/none in last year	1.1 (0.7 – 2.0)	0.6	2.6 (−5.5 – 10.6)	0.5
Number of alcohol drinks a day (Ref. = 1 drink or less)	Former heavy drinker	1.1 (0.4 – 3.2)	0.7	3.0 (−11.0 – 16.9)	0.6
	1 to 2	1.3 (0.9 – 2.1)	0.1	2.1 (−3.4 – 7.5)	0.4
	2 to 4	2.4 (1.1 – 5.0)	0.02	6.1 (−5.0 – 17.2)	0.2
	5 or more	0.9 (0.3 – 2.8)	0.7	9.6 (−3.0 – 22.2)	0.1

[1]Odds ratio calculated by logistic regression for LSM≥ 9.5 kPa.

[2]Beta-coefficient calculated by linear regression for CAP dB/m.

[3]Adjusted model included age, gender, race, vigorous activity, alcohol consumption in the last year, smoking history, BMI, and education level.