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Tea and coffee consumption and the 15-Year risk of cardiovascular events: the Isfahan cohort study (ICS)

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Abstract

Background This study aimed to investigate the association between tea and coffee consumption and the 15-year incidence of cardiovascular events and mortality among the Iranian population.

Methods The present study Data were obtained from the Isfahan Cohort Study (ICS), a prospective cohort study of ≥ 35-year-old healthy adults in central Iran from 2001 to 2017. This study was conducted using baseline data on tea and/or coffee consumption per day/week from ICS to identify the occurrence of any new cardiovascular events, including acute myocardial infarction (AMI), unstable angina (UA), stroke, cardiovascular disease (CVD), sudden cardiac death (SCD), cardiovascular mortality, and all-cause mortality.

Results 5248 participants with complete data were included in the study. After full adjustments, compared to participants with the lowest tea intake, the risk of AMI was significantly higher for participants with the highest tea intake (hazard ratio (HR) = 1.83; 95% confidence interval (CI): 1.10, 3.07; p for trend = 0.060). Also, moderate-tea drinking was associated with a 66% increased risk of AMI compared to the lowest-tea drinking (HR = 1.66; 95%CI: 1.03, 2.70). No significant association was observed between tea consumption and other CVD events or all-cause mortality. Moreover, after full adjustment, no significant association was observed between tea intake above the median and cardiovascular events or all-cause mortality or between coffee consumption and study outcomes.

Conclusions High tea consumption significantly increases the risk of AMI; however, high tea and coffee consumption had no significant association with other cardiovascular events. Future research is needed, especially in Iran and the Middle East, to clarify and evaluate more factors related to the complex nature of tea and coffee consumption and cardiovascular events.

Keywords Tea, Coffee, Cardiovascular diseases, Myocardial infarction, Mortality, Iran

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Background

Cardiovascular diseases (CVD) continue to be the leading cause of mortality globally, significantly impacting overall health and leading to substantial healthcare expenses [1]. The Global Burden of Diseases study estimated the effects of several risk factors on CVD [2]. In 2021, dietary risks were responsible for 6.58 million deaths due to CVDs and 8 million deaths, further confirming the role of nutrition in cardiovascular events [2]. Among the different types of foods and drinks, coffee and tea are the primary sources of caffeine, remaining the most consumed beverages globally after water and in many populations, particularly Asians with a long tradition of consuming tea [3, 4].

Worldwide, people consume an average of 6.3 billion kg of tea annually, with each Iranian drinking approximately 1.5–1.6 kg per year, ranking as one of the highest consumers of tea among countries [5, 6]. However, the Iranian population have less coffee intake than other countries, ranking Iran 133rd out of 161 countries in per capita coffee consumption (about 0.01 kg in 2001, rising to 0.14 kg in 2020) [7]. Over the years, many studies have investigated the role of tea and coffee in various adverse outcomes, especially cardiovascular events and mortality, leading to different heterogeneous results, including harmful, neutral, or favorable effects of these beverages [3, 8–13].

In a pooled analysis of 12 cohort studies conducted in Asian countries, coffee intake significantly reduced the risk of cardiovascular mortality and all-cause death among this population [3]. Coffee intake was also linked to a reduced risk of CVD incidence in a cohort of Iranian people, whereas tea intake increased this risk [14]. However, emerging evidence suggests that in severely hypertensive patients, high coffee intake can be attributed to an increased risk of cardiovascular mortality, but it has no significant effect on participants in other blood pressure (BP) categories [13]. Meanwhile, green tea consumption did not increase cardiovascular mortality risk across all BP categories [13]. Moreover, in a study of the Japanese population, black tea intake showed no significant association with total CVD and stroke, whereas, in a metaanalysis of population-based studies, daily tea intake within a healthy diet reduced the risk of CVD mortality by 4%, cardiovascular events by 2%, and all-cause death by 1.5% [8, 15].

The study results may be inconsistent and not representative of the Iranian population due to differences in tea and coffee consumption cultures, types, and preparation methods. Given the regular consumption of these beverages throughout adulthood, even minor health impacts can significantly affect public health [16]. The Iranian population, characterized by high tea and low coffee intake, necessitates further research on the effects of tea

on cardiovascular events including myocardial infarction (MI), cardiovascular disease (CVD), stroke, sudden cardiac death (SCD), cardiovascular mortality, and all-cause mortality. This cohort study aimed to assess the association between tea and coffee consumption and the 15-year incidence of cardiovascular events and mortality among Iranians using data from the Isfahan Cohort Study (ICS).

Methods

Study design and population

The present study data were obtained from the ICS framework, a population-based, prospective cohort study focusing on healthy and mentally competent adults aged 35 years and older residing in three urban areas: Isfahan, Najaf-Abad, and Arak, situated in central Iran. This research utilized the baseline data collected from the ICS in 2001, which was subsequently monitored every two years for 15 years to identify the occurrence of any new cardiovascular event until 2017. Other variables were reassessed without cardiovascular events in the subsequent 6-year evaluations (2007 and 2013). We did not include pregnants or a history of preexisting CVD, such as MI, stroke, or heart failure as well as inflammatory states like cancers, autoimmune disease, liver and kidney diseases. Comprehensive details regarding the methodology and design of the ICS have been documented in prior publications [17]. Subjects who lacked data on tea and coffee consumption were also excluded from this study. Overall, 5248 participants with complete data were included in the study. Figure 1 illustrates the flow diagram of cohort selection. This study was approved by the ethics committee of Isfahan University of Medical Sciences (IR.MUI.MED.REC.1401.412). Written informed consent was obtained from all patients, and the Declaration of Helsinki was considered.

Data collection

A structured interview was conducted by trained interviewers using a questionnaire to obtain participants' characteristics (age, sex, educational level, and marital status), family history of CVD, and lifestyle-related habits (smoking status and physical activity). Physical activity was examined using the International Physical Activity Questionnaire (IPAQ) [18].

Body mass index (BMI) was computed by dividing the mass in kilograms (kg) by the square of height in meters (kg/m²). Participants were categorized into four BMI groups: underweight (<18.5 kg/m²), normal weight (18.5–24.9 kg/m²), overweight (25–29.9 kg/m²), and obese (\geq 30 kg/m²). Waist circumference (WC) was assessed at the most constricted waist point. WC measurements of \geq 102 centimeters (cm) in males and \geq 88 cm in females indicated abdominal obesity. Systolic BP (SBP) and diastolic BP (DBP) were recorded utilizing

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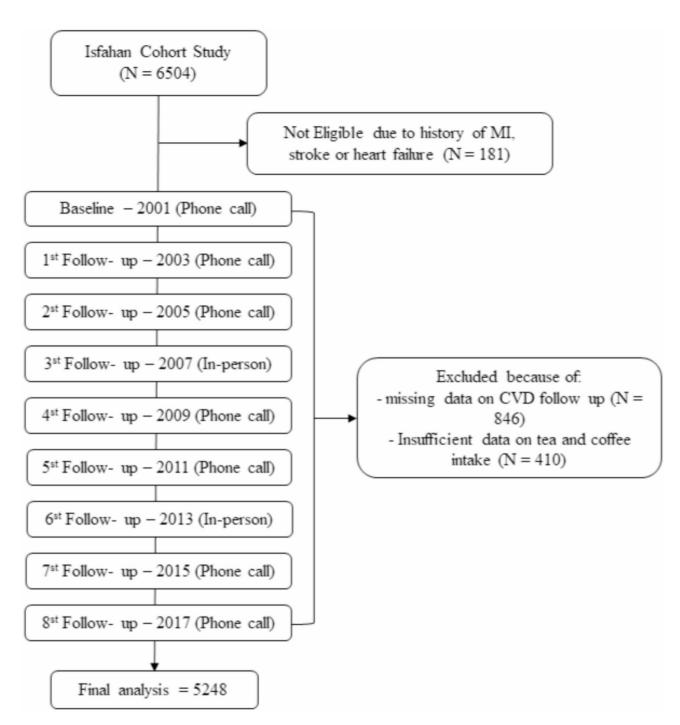


Fig. 1 Flowchart of the study population and data availability in the Isfahan Cohort Study (ICS)

a standard cuff alongside a calibrated sphygmomanometer following a resting period of five minutes. SBP readings of ≥ 140 mmHg or DBP readings of ≥ 90 mmHg, or the administration of anti-hypertensive medications, were classified as hypertension.

Fasting blood samples from all participants were collected and stored at -70 Celsius (°C) after serum separation at the Isfahan Cardiovascular Research Institute. The following parameters were quantitatively assessed in

the participants' blood: fasting blood glucose (FBS), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglyceride (TG). Biochemical variables were evaluated using standard laboratory methods. Diabetes mellitus (DM) was operationally defined as FBS \geq 126 mg/dl or ongoing DM pharmacotherapy. A total cholesterol level \geq 240 mg/dl was regarded as abnormal and indicative of hypercholesterolemia.

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Dietary assessment

Dietary intake data were evaluated utilizing a validated qualitative block format, 48-item food frequency questionnaire (FFQ) [19]. During a face-to-face interview administered by trained dietitians, participants were asked to report their frequency of consumption of each food item over the previous year, responding on a daily, weekly, or monthly basis in an open-ended format. Our analyses computed and employed the average intake of various food items every week [19]. Information related to the consumption of tea and coffee was obtained in the form of two questions in the questionnaire: How many cups/glasses of tea and/or coffee do you drink daily and/ or weekly? The participants were also asked for data regarding the use of caloric sweeteners, such as milk and sugar, added to coffee or tea. Finally, data regarding other food items, including hydrogenated vegetable oil (HVO), non-HVO, cereals, red meat, fish, fruits and vegetables, nuts and seeds, legumes, fast foods, animal fats, sweets, soft drinks, and beverages were collected.

The FFQ employed in this study assessed how often food was consumed in a week (time per week), regardless of portion size. Nevertheless, our validation study revealed that variations in consumption frequency outweighed portion sizes in determining differences among individuals [19]. A dietary intake assessment was performed at baseline in 2001, providing a snapshot of participants' dietary habits during that period.

Ascertainment of cardiovascular events

Two panels, each with four cardiologists and one neurologist, reviewed the cases to confirm related outcomes. CVD was classified into four categories: MI (both fatal and non-fatal), SCD, unstable angina (UA), and stroke (both fatal and non-fatal). MI was diagnosed based on two of the following criteria of the World Health Organization (WHO) Expert Committee: typical chest pain > 30 min, ST-segment elevation > 0.1mV in at least two adjacent leads, and rise in cardiac biomarkers [20]. UA was defined as chest pain lasting at least 20 min, even at rest, or new-onset angina with specific electrocardiogram (ECG) in the ST segment or T waves in at least two adjacent leads [21]. SCD was identified as unexpected death within 24 h of symptom onset without an obvious cause. Stroke was defined as focal neurological symptoms lasting for >24 h. Cardiovascular mortality included any fatalities due to cardiovascular events or SCD, defined as death occurring within an hour of symptom onset, during an observed cardiac arrest, or from a sudden collapse without prior symptoms lasting > 41 h. Additional details were gathered from the hospital records if needed, and for every CVD case, a control without CVD but with similar risk factors was selected. The panel independently made final decisions on outcomes, although they considered the diagnoses made by in-hospital clinicians. Details regarding the follow-up process have been provided in a previous publication [22].

Statistical analysis

Continuous data were reported as mean ± standard deviation (SD), and categorical data were reported as numbers (percentages). Comparisons of variables across different levels of tea consumption were conducted using analysis of variance (ANOVA) for continuous variables and the chi-square test for categorical variables. Two independent sample t-tests and chi-square tests were used to compare the coffee drinker and non-drinker groups. Time to cardiovascular events and all-cause mortality was defined as the time from enrolment to the occurrence of an event, loss to follow-up, or end of follow-up, whichever occurred first. The association between coffee and tea consumption and the risk of cardiovascular events and all-cause mortality was investigated by the Cox proportional hazard model. Several potential confounders were adjusted in the modeling process through four hierarchical steps in the entire population. First, a crude model was performed without adjustment for covariates. Model 1 was adjusted for age (year) and sex (male/female). Model 2 was additionally adjusted for education (0-5 years / 6-12 years / >12 years), smoking status (never/ever smoking), physical activity (METSminute/day), DM (yes/no), hypertension (yes/no), hypercholesterolemia (yes/no), family history of CVD (yes/ no) and BMI (kg/m²). Finally, model 3 was additionally adjusted for HVO (time/week), non-HVO (time/ week), cereals (time/week), red meat (time/week), fish (time/week), fruits and vegetables (time/week), nuts and seeds(time/week), legumes (time/week), fast foods (time/ week), animal fats (time/week), sweets (time/week), soft drinks (time/week), beverages (time/week), sugar (time/ week), and milk (time/week). Schoenfeld's global test of residuals was used to evaluate the proportional hazard assumption of the multivariable Cox model. The *p*-values for the linear trend analysis were obtained by scoring the tea consumption tertiles from 1 to 3, corresponding to the lowest to highest category, entering as a continuous variable in the model. However, because of the sparsity of the data, only the crude model and model 1 were used for the coffee groups. All reported p values were considered significant at p < 0.05. Statistical analysis was conducted with a 5% error using SPSS for Windows version 25 (SPSS Inc., Chicago, IL, USA).

Results

Among the 5248 participants enrolled in the current study, 48.0% were male, and the mean \pm SD age was 50.74 ± 11.65 . Of these, 81 participants (1.54%) were coffee drinkers, and 1472 (28.05%) were the highest tea

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drinkers. The baseline participant characteristics of tea and coffee consumption are shown in Table 1. The mean intake of tea was 5.64 ± 2.89 cups per week in the total population, and the mean intake of coffee was 1.73 ± 1.41 cups per week in the coffee-drinker population. Participants who did not drink coffee were significantly older than coffee drinkers (50.80 ± 11.68 vs. 47.01 ± 8.75 ; p = 0.012). Males and married participants tended to drink more tea (p < 0.001). Higher education was associated with both low tea intakes and coffee drinker group (p < 0.001).

The proportion of smokers was directly associated with tea consumption levels (p<0.001). Coffee drinkers were more physically active than non-drinkers (1029.89 ± 583.16 vs. 864.74 ± 543.80 MET-minute/day; p=0.007) (Table 1). Similarly, a higher tea intake

was positively associated with physical activity levels (p < 0.001) (Table 1).

Higher tea intake was significantly associated with lower BMI, WC, SBP, DBP, and TC levels (p<0.05) (Table 1). The mean HDL-C levels across the tertiles of tea consumption were significantly different (p=0.006). The prevalence of abdominal obesity and hypertension was significantly lower in the highest-tea intake consumers (p<0.05) (Table 1). No differences were reported in terms of FBS, LDL-C, TG, DM, hypercholesterolemia, and family history of CVD across the tertiles of tea consumption and coffee drinking groups (Table 1). Moreover, the baseline characteristics and dietary intake based on the CVD incidence and median tea intake are also illustrated in Table S1-S4.

The dietary intake of the study participants in the tea consumption tertiles and coffee drinker and non-drinker

Table 1 Baseline characteristics of study participants based on the tertiles of tea and coffee intake

Variable	Tea intake			P	Coffee intake	Coffee intake	
	T1 (n = 1434)	T2 (n = 2342)	T3 (1472)		No (n=5167)	Yes (n = 81)	
Tea intake (cups/week)	2.58 ± 0.64	5.11±0.87	9.49 ± 1.95	< 0.001	5.65 ± 2.89	4.98 ± 3.02	0.036
Coffee intake (cups//week)	1.61 ± 1.10	2.20 ± 2.02	1.35 ± 0.75	0.107	0.00 ± 0.00	1.73 ± 1.41	
Age, years (mean ± SD)	50.67 ± 11.81	50.90 ± 11.72	50.56 ± 11.39	0.699	50.80 ± 11.68	47.01 ± 8.75	0.012
Sex (Male), n (%)	570 (39.7)	1099 (46.9)	852 (57.9)	< 0.001	2482 (48.0)	39 (48.1)	0.984
Married, n (%)	1305 (91.0)	2111 (90.1)	1364 (92.7)	0.028	4706 (91.1)	74 (91.4)	0.930
Education, n (%)				< 0.001			< 0.001
0–5 years	972 (67.9)	1653 (70.7)	1113 (75.7)		3699 (71.7)	39 (48.1)	
6–12 years	368 (25.7)	529 (22.6)	283 (19.2)		1147 (22.2)	33 (40.7)	
>12 years	91 (6.4)	156 (6.7)	75 (5.1)		313 (6.1)	9 (11.1)	
Smoking status, n (%)				< 0.001			0.859
Never	1215 (84.7)	1877 (80.1)	1032 (70.1)		4061 (78.6)	63 (77.8)	
Ever smoking	219 (15.3)	465 (19.9)	440 (29.9)		1106 (21.4)	18 (22.2)	
Physical activity (METs-minute/day)	808.46 ± 524.35	856.81 ± 536.78	930.75 ± 569.76	< 0.001	864.74 ± 543.80	1029.89 ± 583.16	0.007
BMI (kg/m²)	27.28 ± 4.63	26.87 ± 4.62	26.28 ± 4.66	< 0.001	26.81 ± 4.66	27.07 ± 4.38	0.756
WC (cm)	95.82 ± 12.02	95.04 ± 12.17	93.44 ± 12.39	< 0.001	94.81 ± 12.24	94.37 ± 11.30	0.692
SBP (mmHg)	89.53 ± 33.37	88.36 ± 32.48	87.91 ± 32.33	0.001	121.75 ± 20.93	121.16 ± 22.59	0.616
DBP (mmHg)	79.09 ± 12.06	78.74±11.35	77.49 ± 11.25	< 0.001	78.49 ± 11.55	77.94 ± 10.58	0.533
FBG (mg/dl)	89.53 ± 33.37	88.36 ± 32.48	87.91 ± 32.33	0.454	88.57 ± 32.71	87.46 ± 31.05	0.856
TC (mg/dl)	215.37 ± 50.42	212.33 ± 49.24	209.89 ± 51.62	0.026	212.48 ± 50.23	211.80 ± 53.39	0.587
LDL-C (mg/dl)	130.07 ± 42.74	128.57 ± 42.16	126.70 ± 42.64	0.137	128.44 ± 42.38	128.88 ± 47.53	0.616
HDL-C (mg/dl)	47.73 ± 10.70	46.60 ± 10.39	46.70 ± 10.27	0.006	46.91 ± 10.46	48.30 ± 10.13	0.202
TG (mg/dl)	198.59 ± 127.77	194.54 ± 116.32	188.02 ± 116.30	0.075	193.78 ± 119.08	195.94 ± 149.73	0.609
Weight status, n (%)				< 0.001			0.568
Underweight (BMI < 18.5)	17 (1.2)	44 (1.9)	35 (2.4)		96 (1.9)	0 (0)	
Normal (BMI = 18.5-24.99)	446 (31.3)	795 (34.2)	582 (39.9)		1792 (35.0)	31 (38.3)	
Overweight (BMI = 25-29.99)	594 (41.7)	971 (41.8)	561 (38.5)		2092 (40.8)	34 (42.0)	
Obesity (BMI≥30)	366 (25.7)	512 (22.0)	280 (19.2)		1142 (22.3)	16 (19.8)	
Abdominal obesity, n (%)	789 (55.2)	1160 (49.8)	596 (40.6)	< 0.001	2508 (48.7)	37 (45.7)	0.587
Hypertension, n (%)	438 (30.5)	668 (28.5)	357 (24.3)	0.001	1442 (27.9)	21 (25.9)	0.693
Diabetes Mellitus, n (%)	141 (9.8)	193 (8.2)	110 (7.5)	0.064	438 (8.5)	6 (7.4)	0.731
Hypercholesterolemia, n (%)	504 (35.1)	767 (32.7)	462 (31.4)	0.091	1706 (33.0)	27 (33.3)	0.952
Family history of CVD, n (%)	172 (12.5)	283 (12.7)	150 (10.7)	0.193	596 (12.1)	9 (11.3)	0.819

Abbreviations: T=tertile; P=p-value for trend; BMI=body mass index; WC=waist circumference; SBP=systolic blood pressure; DBP=diastolic blood pressure; FBG=fasting blood glucose; TC=total-cholesterol; LDL-C=low-density lipoprotein-cholesterol

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groups are demonstrated in Table 2. Higher tea intake was significantly associated with higher HVO, cereal, red meat, legume, SSB, sugar, and milk intakes (all p < 0.05). In contrast, the intake of non-HVO, fish, and fruits and vegetables decreased considerably with increasing tea intake (all p < 0.05). Coffee drinking was positively associated with higher intake of non-HVO, fish, fruit and vegetables, nuts and seeds, fast food, animal fat, and SSB (p < 0.05).

During the follow-up period, the incidence rates of AMI, UA, stroke, CVD, SCD, CVD mortality, and allcause mortality were 2.95%, 6.92%, 3.18%, 14.81%, 1.75%, 3.28%, and 9.03%, respectively. The risk of cardiovascular events according to tea and coffee consumption is presented in Table 3. Tea intake was significantly associated with the risk of AMI in the both crude and adjusted models. After full adjustment, the risk of AMI was significantly 83% higher in participants with the highest tea intake than in those with the lowest tea intake (hazard ratio (HR) = 1.83; 95% confidence interval (CI): 1.10, 3.07; p for trend = 0.060). Also, moderate-tea drinking was associated with a 66% increased risk of AMI compared to the lowest-tea drinking (HR = 1.66; 95%CI: 1.03, 2.70). No significant association was observed between tea consumption and UA, stroke, CVD, SCD, CVD mortality, and all-cause mortality was observed (all *p*-values > 0.05) (Table 3). There was no considerable association between coffee drinking and cardiovascular events and all-cause mortality based on both crude and adjusted models (all p-values > 0.05) (Table 3). When stratified by the median tea intake, > median tea intake was significantly associated with a 42% higher risk of AMI in the crude model (HR = 1.42; 95%CI: 1.03, 1.94); however, the association was insignificant after full adjustment (HR = 1.27; 95%CI: 0.89, 1.81). Furthermore, no significant association was found between > median tea intake and other cardiovascular events, such as UA, stroke, CVD, SCD, CVD mortality, and all-cause mortality (all p > 0.05) (Table S5).

Discussion

In this cohort study, we observed a significant positive association between high tea consumption and AMI risk. However, no other significant association was revealed between tea and coffee consumption and the risk of other cardiovascular events, including UA, stroke, CVD, SCD, CVD mortality, and all-cause mortality. To date, several studies have evaluated the effect of different types of tea and coffee on the risk of cardiovascular events; however, after decades, there are still controversial results in different countries, indicating that there still needs to be more clarity regarding this topic. Also, few long-term studies have established black or green tea as a primary CVD prevention [23].

Unlike most studies in the literature search, our study found that a higher tea intake did not have a cardiovascular protective relationship; instead, it was associated with an increased risk of AMI by 83% in Iranian participants. However, no significant associations were observed with other cardiovascular outcomes. One possible reason for the increased risk of AMI with tea consumption in our study can be explained by excessive tea intake in the Iranian population. A recent meta-analysis by Yang et al. comprised 24 studies on green tea and 11 studies on black tea consumption [24]. The results showed that consuming less than four cups of black tea per day may successfully prevent coronary artery disease (CAD), but consuming more than 4–6 cups per day may increase the risk of CAD. Moreover, other studies demonstrated

Table 2 Dietary intake of study participants across tea tertiles and coffee intake

Food intake (time per week)	Tea intake		Coffee intake				
	T1 (n=1434)	T2 (n = 2342)	T3 (n = 1472)	Р	No (n=5167)	Yes (n = 81)	Р
Tea intake (cups/week)	2.58 ± 0.64	5.11 ± 0.87	9.49 ± 1.95	< 0.001	5.65 ± 2.89	4.98 ± 3.02	0.036
Coffee intake (cups/week)	1.61 ± 1.10	2.20 ± 2.02	1.35 ± 0.75	0.107	0.00 ± 0.00	1.73 ± 1.41	
HVO	7.46 ± 4.96	8.08 ± 4.85	8.83 ± 4.77	< 0.001	8.11 ± 4.88	8.75 ± 5.32	0.369
Non-HVO	3.19 ± 4.26	2.52 ± 3.85	1.73 ± 3.11	< 0.001	2.45 ± 3.80	4.39 ± 4.62	< 0.001
Cereals	26.79 ± 8.07	27.46 ± 6.91	27.76 ± 6.89	0.001	27.38 ± 7.23	26.32 ± 8.19	0.422
Red meat	3.87 ± 2.66	4.22 ± 2.79	4.37 ± 2.71	< 0.001	4.16 ± 2.73	4.44 ± 2.93	0.471
Fish	0.54 ± 0.95	0.43 ± 0.87	0.33 ± 0.68	< 0.001	0.43 ± 0.85	0.77 ± 0.86	< 0.001
Fruits and vegetables	13.91 ± 7.71	13.23 ± 7.00	12.37 ± 6.70	< 0.001	13.14±7.13	15.47 ± 7.40	0.003
Nut and seeds	1.18 ± 2.30	1.09 ± 2.11	1.14 ± 2.25	0.850	1.12 ± 2.20	1.83 ± 2.56	< 0.001
Legumes	3.34 ± 2.74	3.45 ± 2.44	3.81 ± 2.55	< 0.001	3.51 ± 2.55	4.23 ± 3.29	0.146
Fast foods	0.60 ± 1.16	0.54 ± 0.99	0.54 ± 1.13	0.068	0.55 ± 1.07	0.98 ± 1.47	< 0.001
Animal fats	1.95 ± 2.86	1.84 ± 2.80	1.67 ± 2.94	0.309	1.89 ± 2.84	3.06 ± 3.47	< 0.001
SSB	1.28 ± 1.86	1.25 ± 1.76	1.35 ± 1.75	0.006	1.28 ± 1.78	1.98 ± 2.08	< 0.001
Sugar	5.47 ± 2.45	5.70 ± 2.15	5.85 ± 2.25	< 0.001	5.68 ± 2.26	5.91 ± 2.78	0.663
Milk	0.59 ± 1.76	0.65 ± 1.69	0.80 ± 1.81	0.003	0.67 ± 1.73	1.09 ± 2.51	0.134

 $\textbf{Abbreviations:} \ Q = \text{quartile;} \ P = p \text{-value;} \ HVO = \text{hydrogenated vegetable oil;} \ SSB = \text{sweets, soft drink, and beverages}$

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Table 3 Multiple adjusted hazard ratios of tea and coffee intake for cardiovascular events

Events rate	rate	Person-years	Tea intake			P trend	Coffee intake		Р
	ŕ	T1 (n = 1434)	T2 (n = 2342)	T3 (n = 1472)	_	No (n=5167)	Yes (n = 81)	—	
		2.58±0.64	5.11±0.87	9.49 ± 1.95		0.00 ± 0.00	1.73 ± 1.41		
AMI									
Crude model	155	60365.40	1	1.85 (1.17, 2.91)	2.32 (1.45, 3.72)	0.001	1	0.448 (0.063-3.198)	0.42
Model 1 ^a	155		1	1.74 (1.10, 2.74)	2.09 (1.30, 3.36)	0.007	1	0.608 (0.085-4.357)	0.62
Model 2 ^b	146		1	1.88 (1.17, 3.02)	2.29 (1.39, 3.78)	0.004			
Model 3 ^c	133		1	1.66 (1.03, 2.70)	1.83 (1.10, 3.07)	0.060			
Unstable Angina									
Crude model	363	59988.46	1	1.24 (0.97, 1.58)	0.96 (0.72, 1.29)	0.427	1	1.429 (0.676-3.019)	0.350
Model 1	363		1	1.21 (0.95, 1.55)	0.95 (0.71, 1.27)	0.381	1	1.866 (0.881-3.953)	0.104
Model 2	338		1	1.21 (0.93, 1.56)	0.91 (0.67, 1.24)	0.286			
Model 3	322		1	1.28 (0.98, 1.68)	1.02 (0.73, 1.41)	0.667			
Stroke									
Crude model	167	60306.30	1	0.72 (0.50, 1.03)	0.86 (0.58, 1.26)	0.711	1	0.415 (0.058-2.960)	0.380
Model 1 ^a	167		1	0.69 (0.48, 0.99)	0.85 (0.57, 1.26)	0.737	1	0.636 (0.089-4.556)	0.652
Model 2 ^b	153		1	0.79 (0.54, 1.14)	0.77 (0.50, 1.18)	0.312			
Model 3 ^c	144		1	0.79 (0.53, 1.15)	0.74 (0.47, 1.15)	0.243			
CVD									
Crude model	777	59257.91	1	1.13 (0.95, 1.35)	1.12 (0.92, 1.35)	0.409	1	1.106 (0.625-1.956)	0.730
Model 1 ^a	777		1	1.09 (0.92, 1.30)	1.07 (0.88, 1.30)	0.679	1	1.592 (0.898-2.821)	0.11
Model 2 ^b	716		1	1.15 (0.96, 1.38)	1.06 (0.87, 1.31)	0.876			
Model 3 ^c	662		1	1.16 (0.96, 1.40)	1.04 (0.84, 1.30)	0.951			
SCD									
Crude model	92	60694.75	1	0.84 (0.57, 1.54)	1.03 (0.60, 1.77)	0.820	1	2.268 (0.718–7.167)	0.163
Model 1 ^a	92		1	0.86 (0.53, 1.42)	0.93 (0.54, 1.60)	0.920			
Model 2 ^b	79		1	0.92 (0.53, 1.58)	1.06 (0.59, 1.91)	0.730			
Model 3 ^c	69		1	1.03 (0.57, 1.87)	1.02 (0.53, 1.95)	0.984			
CVD mortality									
Crude model	172	60694.75	1	0.89 (0.62, 1.28)	1.07 (0.73, 1.58)	0.535	1	1.606 (0.596-4.329)	0.349
Model 1 ^a	172		1	0.84 (0.58, 1.20)	1.02 (0.69, 1.51)	0.635			
Model 2 ^b	149		1	1.00 (0.67, 1.50)	1.20 (0.78, 1.85)	0.334			
Model 3 ^c	134		1	1.06 (0.70, 1.62)	1.16 (0.73, 1.85)	0.523			
All-cause mortality	,								
Crude model	474	60694.75	1	0.93 (0.75, 1.16)	0.93 (0.73, 1.18)	0.641	1	0.870 (0.389-1.946)	0.73
Model 1 ^a	474		1	0.88 (0.71, 1.10)	0.90 (0.71, 1.15)	0.567	1	1.753 (0.780-3.939)	0.17
Model 2 ^b	422		1	0.94 (0.74, 1.18)	0.89 (0.69, 1.16)	0.428			
Model 3 ^c	393		1	0.94 (0.74, 1.20)	0.86 (0.65, 1.13)	0.266			

Abbreviations: P=Pvalue; T=Tertile; AMI=acute myocardial infarction; SCD=sudden cardiac death; CVD=cardiovascular disease. **a**: adjusted for age (year) and sex (male/ female). **b**: Additionally, adjusted for education (0–5 years / 6–12 years / >12years), smoking status (never / ever smoking), physical activity (METS-minute/day), diabetes mellitus (yes/ no), hypertension (yes/ no), hypercholesterolemia (yes/ no), family history of cardiovascular disease (yes/ no), and body mass index (kg/m²). **c**: Additionally, adjusted for hydrogenated vegetable oil (time/week), non-hydrogenated vegetable oil (time/week), cereals (time/week), red meat (time/week), fish (time/week), fruits and vegetables (time/week), nut and seeds (time/week), legumes (time/week), fast foods (time/week), animal fats (time/week), sweets (time/week), soft drinks (time/week), beverages (time/week), sugar (time/week), and milk (time/week)

a beneficial effect of 1–3 cups/day of tea consumption on CVD prevention [9]. In a study by Hao et al. on 5856 Chinese participants, participants who drank tea > 3 cups/day had a 29% higher risk of AMI than tea non-drinkers, and a similar trend with a higher risk of 73% was found in green tea drinkers who drank more than 3 cups/day compared with tea non-drinkers [25].

In the subgroup analysis by continent in the metaanalysis study by Yang et al., a significant negative correlation of 8% was reported between green tea intake and the risk of CAD in the Asian population; however, this correlation was not significant in the Western Europe/ Oceania population [24]. In line with our results, two possible factors are race and high tea intake, which may affect cardiovascular outcomes; consumption of black tea is more common than green tea consumption in Western societies and Iran. Unfortunately, we failed to gather data regarding the type of tea and its preparation, which might have impacted the outcomes. However, previous research on the Iranian population in 2009 (more

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similar to population habits at that time) indicated that the majority (96.8%) of the Iranian population consumed black tea on a daily basis background [26]. In contrast, only a small number (5.8%) of all participants consumed green tea every day [26]. Notably, the majority of these green tea drinkers were of a particular minority ethnic background (Turkmen) [26].

The manner in which the leaves are treated determines the kind of tea made from them. Green tea is made from unfermented leaves, but black tea is made from fermented leaves [25, 27]. Black tea production involves oxidation, which may transform flavonoid-like catechins in green tea into more complicated forms. This could explain why black and green teas have distinct benefits. Previous research has indicated that tea catechins may reduce the risk of CVD, which can also be provided by other sources such as cocoa, berries, apples, onions, broccoli, and green vegetables, and not only in tea [25, 27, 28]. A study including several observational studies and five meta-analyses found more evidence for green tea than black tea with different flavonoid profiles, making it challenging to compare this evidence because the populations studied in each of the individual studies on these two types of tea differ significantly in terms of their baseline CVD risks, with few studies on non-Asian populations offering evidence for green tea [29].

In a study by Gaeini et al. in Iran, during a median follow-up period of six years, the risk of CVD was 2.4-fold higher in the highest tertile of tea consumption, which confirms our results regarding the high consumption of tea with cardiovascular events in Iran [14]. One of the reasons proposed in this study is the adverse effects of high sugar intake alongside tea intake in Iran, which has been mentioned in many studies. However, sugar was adjusted for in our study, and other factors should also be considered. Another factor alongside tea that was discussed in previous studies on the protective relationship between tea consumption and CVD risk findings is that tea drinkers may have a healthier lifestyle. For instance, the Dutch and Boston Area Health Study reported that tea intake was associated with a healthier lifestyle and higher social class [30]. However, this point does not apply in our study, as higher education was associated with a low intake of tea.

Fermented tea variants or tea types may affect cardiovascular events differently. Different types of tea are characterized by variations in the types and concentrations of bioactive compounds, and even among the same variety, discrepancies are evident; the phenolic content and antioxidant activity of black, green, and herbal or berry teas may fluctuate by more than two-fold based on prior research findings [31]. Furthermore, the method of tea preparation significantly affects tea contents, with black tea essentially constituting a fermented version of green tea [32].

Tea may contain a variety of additives or artificial colors. In a study by Pouretedal et al., 40 black tea samples were analyzed for aflatoxin contamination, and the results revealed that 30 of the 40 samples were contaminated with aflatoxins [33]. In addition, some studies have mentioned that high levels of some metals in Iranian tea can increase the blood levels of these metals above the normal level when consumed with other foods [34]. Finally, the negative impact of caffeine on the likelihood of cardiovascular events is more likely to be associated with high tea consumption than with low coffee consumption, as tea consumption is the main dietary source of caffeine for Iranians and our demographic [14].

Regular mild-to-moderate coffee drinking has been linked to a lower risk of atrial fibrillation, stroke, CVD, hypertension, heart failure, and all-cause mortality, according to recent research [9, 35]. However, the results of earlier prospective research on the association between coffee consumption and the risk of cardiovascular events, specifically the risk of coronary heart disease (CHD), have been inconsistent. A meta-analysis of 32 prospective cohort studies conducted in 2021 found no evidence of a significant correlation between coffee consumption and CHD incidence [10]. In addition, the type of coffee and its preparation method, such as boiled, unfiltered, or filtered coffee, are important factors that should be evaluated in our study. In a cohort study of 626 patients with CVD in 2022, the findings indicated that individuals with CVD who drank more than four cups of coffee, caffeine, iced tea, or hot tea daily had a higher chance of all-cause mortality [36]. While some studies have found no direct link between coffee consumption and CVD, and some studies suggest that high coffee consumption may elevate the risk of specific cardiovascular events, the low prevalence of coffee consumption among our study participants limits the ability to draw definitive conclusions.

Limitations and future research directions

The results of our investigation must be interpreted considering certain limitations. Initially, the dataset was limited to Persian participants in central Iran; hence, the findings may not be extrapolated to other ethnic groups, necessitating investigations involving more heterogeneous samples. Consequently, it is recommended that subsequent research be conducted within Iranian and Middle Eastern contexts, which have different ethnicities, in addition to Asian societies. Second, in this study, the type, brand, and method of tea and coffee preparation should be considered, which can be one of the reasons for differentiating between the protective and adverse effects of tea. Therefore, these factors should be

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investigated further in future studies. It would be better to examine the ingredients of tea and coffee, including additives or artificial colors, aflatoxin, and metals, along with their effects on cardiovascular events. Third, dietary intake data in this study were collected using a validated qualitative FFQ that assessed food consumption in terms of frequency (times per week) rather than precise quantities (grams or milliliters). Finally, due to the self-reporting nature of the questionnaires, the potential for both recall and reporting bias existed during the data collection process.

Conclusions

In summary, the findings of this cohort study revealed a significant association between high tea consumption and an increased risk of AMI, contrary to various studies that suggest the potential cardiovascular benefits of tea. However, no significant associations were found between tea or coffee consumption and other cardiovascular outcomes, including UA, stroke, CVD, SCD, cardiovascular mortality, and all-cause mortality. These findings highlight the complexity of the association between tea and coffee consumption and cardiovascular events, suggesting that differences in tea consumption, including cultural habits, preparation methods, ethnicity, type of tea and coffee, and possibly the presence of additives, may play a crucial role in cardiovascular outcomes. These results underscore the need for further research, particularly in Middle Eastern and Asian populations, to clarify the effects of different types of tea and coffee, preparation methods, and the potential influence of additives on cardiovascular events.

Abbreviations

CVD Cardiovascular disease BP Blood pressure MI Myocardial infarction SCD Sudden cardiac death ICS Isfahan cohort study

IPAQ International physical activity questionnaire

BMI Body mass index
WC Waist circumference
SBP Systolic blood pressure
DBP Diastolic blood pressure
FBS Fasting blood glucose
TC Total cholesterol

LDL-C Low-density lipoprotein cholesterol HDL-C High-density lipoprotein cholesterol

TG Triglyceride
DM Diabetes mellitus
FFQ Food frequency questionnaire
UA Unstable angina
WHO World health organization
ECG Electrocardiogram
SD Standard deviation

HVO Hydrogenated vegetable oil
CI Confidence interval
HR Hazard ratio

CAD Coronary artery disease
CHD Coronary heart disease

Supplementary Information

The online version contains supplementary material available at https://doi.or q/10.1186/s12937-025-01093-w.

Supplementary Material 1

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Author contributions

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The research ethics committee of Isfahan University of Medical Sciences approved the study protocol (IR.MUI.MED.REC.1401.412). All participants provided informed written consent to participate in the study.

Consent for publication

Written and informed consent to publish this information is obtained from the patient.

Competing interests

The authors declare no competing interests.

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