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Cigarette smoking and cardiovascular disease incidence and all-cause mortality: the modifying role of diet quality

Mostafa Norouzzadeh[1], Farshad Teymoori[1,2], Hossein Farhadnejad[2], Nazanin Moslehi[2], Parvin Mirmiran[2*], Seyedeh Tayebeh Rahideh[1*] and Fereidoun Azizi[3]

Abstract

Background This study examines the potential long-term joint association between smoking and diet quality as modifiable risk factors concerning cardiovascular diseases (CVDs) incidence and all-cause mortality among current and former smokers.

Methods The study followed 955 smokers from the third and fourth examinations of the Tehran Lipid and Glucose Study to March 2018. Dietary data was collected using a food frequency questionnaire. Three diet quality indices (DQIs) were computed at baseline: DQI-international (DQI-I), DQI-revised (DQI-R), and Mediterranean-DQI (Med-DQI). Cox proportional hazards regression models were used to determine the HR (95% CI) of the joint association between smoking and diet quality among heavy and light smokers, based on the number of cigarettes per day and pack-years, as well as between current and former smokers based on smoking habits.

Results Over a follow-up period of almost eight years, 94 cases of CVDs (9.80%) and 40 cases of mortality (4.20%) were documented. The lower diet quality based on the Med-DQI was associated with a higher risk of mortality among current smokers (HR:3.45; 95%CI:1.12, 10.57). Light smokers with good diet quality, compared to heavy smokers with poor diet quality, had a lower risk of CVDs incident (HR:0.35; 95%CI: 0.15, 0.83) and all-cause mortality (HR:0.20; 95%CI:0.05, 0.77). Current smokers with good DQI had a lower risk of mortality compared to current smokers with poor DQI (HR:0.26; 95%CI:0.08, 0.80). However, this lower risk was more significant in former smokers with good DQI (HR:0.10; 95%CI:0.02, 0.45).

Conclusions Light and former smokers had a lower risk of developing CVDs and experiencing mortality. However, when coupled with a high-quality diet, this protective effect is even more pronounced.

Keywords Cardiovascular diseases, Mortality, Mediterranean diet, Smoking, Adults

*Correspondence:
Parvin Mirmiran
mirmiran@endocrine.ac.ir
Seyedeh Tayebeh Rahideh
tayebeh_rahideh@yahoo.com; rahide.t@iums.ac.ir
[1]Department of Nutrition, School of Public Health, Iran University of Medical Sciences, Tehran, Iran
[2]Nutrition and Endocrine Research Center, Research Institute for Endocrine Sciences, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[3]Endocrine Research Center, Research Institute for Endocrine Sciences, Shahid Beheshti University of Medical Sciences, Tehran, Iran

Background

Cardiovascular diseases (CVDs) are currently the primary cause of death worldwide [1]. Meanwhile, Iran is also experiencing an upward trend in CVDs incidence [2]. CVDs are multifactorial conditions that result from the interaction of genetic, metabolic, and environmental factors [3, 4]. Smoking and poor diet are among the modifiable and greatest contributors to the global burden of CVDs [5]. The total number of smokers has increased globally, leading to almost 8 million deaths and 200 million disability-adjusted life years (DALYs) in 2019 [6]. Notably, poor diet is responsible for 10.9 million deaths, and 255 million DALYs annually [7].

Smoking and poor diet share common disease-causing mechanisms that can interact and increase the risk of CVDs over time [8]. Smokers often adopt unhealthy dietary habits, which are characterized by lower intake of fiber and higher consumption of fats and sugar [9, 10]. Empirical evidence suggests that smokers with inadequate nutritional status are at a greater risk of developing CVDs, than smokers with healthier diets [9, 11]. However, previous studies mainly focused on individual dietary components, instead of assessment of overall dietary pattern [12–14]. Current evidence suggests that the impact of the entire diet on health outcomes is believed to be greater than that of individual dietary constituents [15, 16]. The Diet Quality Index (DQI) is a well-suited tool to assess dietary patterns and link dietary habits with diseases [17, 18]. Adherence to the DQI has been associated with a lower risk of CVDs, hypertension, and metabolic syndrome [19–21].

According to our literature review, very limited data are available concerning the dietary quality of smokers and its association with the incidence of CVDs and all-cause mortality. Additionally, no study investigates the joint association between smoking and diet quality among smokers, particularly in the Middle East and North Africa (MENA) region. Notably, previous studies have limitations since they did not consider the duration and intensity of smoking [3, 13, 22].

Considering the limited available data, this study aims to evaluate whether a smoker's higher diet quality, either by itself or in combination with lower smoking intensity and duration or quitting smoking, can have an impact on their clinical outcomes. Hence, we examined the joint association between smoking intensity, smoking duration, and diet quality concerning the incidence of CVDs and all-cause mortality.

Methods

Study population

The Tehran lipid and glucose study (TLGS) is an ongoing population-based study aimed at identifying non-communicable disease risk factors and promoting better lifestyles, with six follow-up examinations completed since 1999. The TLGS cohort consists of 15,005 participants aged 3 or more who undergo standardized physical exams, laboratory tests, and medical history updates every 3 years [23].

Since the collection of dietary data started from third examination, participants with complete dietary data on the third examination of TLGS and the new entries participants in the fourth examination were considered as baseline examinations and were followed until the end of sixth examination. In the third survey of the TLGS (2006–08), of 12 523 participants, 3686 were randomly selected for dietary assessment, and in the fourth survey (2009–2011), 7956 randomly selected subjects, agreed to complete dietary assessment. If a participant in the third examination had underreported or overreported energy intake (lower than 800 kcal/d and higher than 4200 kcal/d, respectively) ($n=233$), was excluded but if their energy intake was in the normal range in the fourth examination ($n=100$), their dietary intake in the fourth examination was included as their baseline dietary data. Finally, after excluding those with overunder reports of energy intake in the fourth examination ($n=502$), 8914 participants with complete dietary data were included.

For the present study, of 8914 individuals, participants aged < 30 years ($n=3382$), never smokers ($n=4093$) or participants with any type of CVDs ($n=338$), cancer history ($n=32$), pregnant and lactating women ($n=70$) and missing data ($n=70$) were excluded. Some of them may fall into more than one category. Finally, 955 smokers, free of CVDs entered into the study (Fig. 1) and were followed up until 20 March 2018. During the follow-up, all participants were assessed for any type of CVDs, and the mortality data were recorded.

Dietary assessment

Skilled dieticians collected dietary data using a reliable and validated semiquantitative food frequency questionnaire (FFQ) comprising 168 items [24]. The study involved collecting information about the consumption frequency of various food items over the past year, categorized as daily, weekly, or monthly. The reported portion sizes of the consumed foods were converted from household measures to grams for analysis purposes. The energy and nutrient content of the food was calculated by utilizing the United States Department of Agriculture (USDA) food composition table (FCT) [25]. For local food items that were not listed in the USDA FCT, the Iranian FCT [26] was used.

Demographic and clinical measurements

Trained interviewers conducted face-to-face interviews with participants using a standardized questionnaire to collect demographic variables. A telephone follow-up

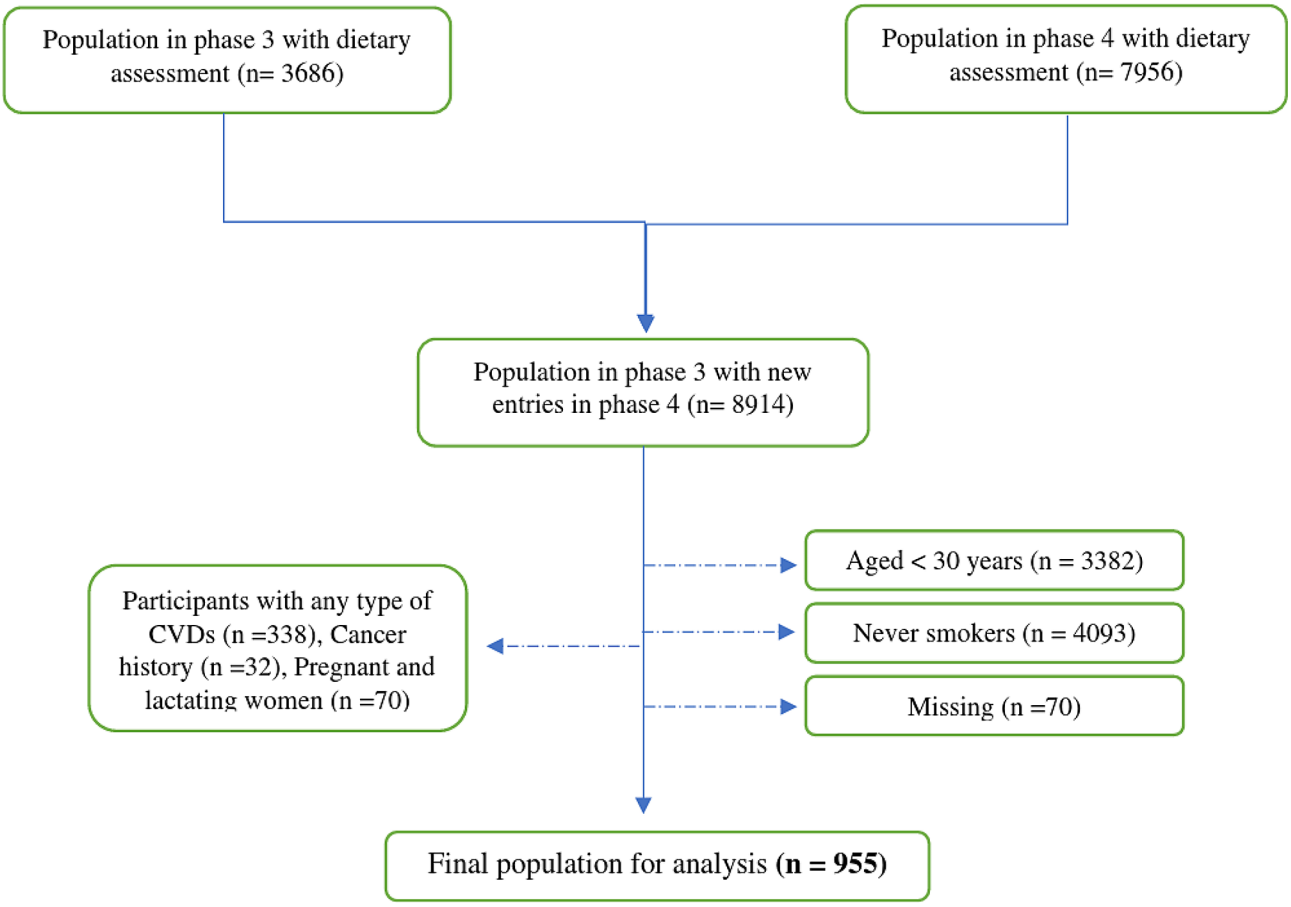

Fig. 1 Diagram of the follow-up participants

was performed annually to check for new medical events, with additional information gathered by a physician through a home visit or by accessing medical records if needed.

The validated Modifiable Activity Questionnaire (MAQ) was used to assess physical activity [27]. The MAQ questionnaire is divided into two categories based on recreational and occupational activities. Total physical activity was measured as metabolic equivalent minutes per week (MET-min/week) based on the frequency and duration of each activity over the preceding year.

Participants were asked to remain seated for 15 min before their blood pressure was measured. A qualified physician then took blood pressure readings twice, with at least a 30-second interval between measurements. A standard mercury sphygmomanometer was used for this purpose, which was calibrated by the Iranian Institute of Standards and Industrial Research. The mean of the two readings was recorded as the participant's blood pressure. Height and weight were measured using a standard protocol. Participants were instructed to remove their shoes and wear light clothing. Height was measured using a stadiometer, while weight was measured using a calibrated weighing scale.

Serum glucose concentration was measured by drawing a blood sample between 7:00 and 9:00 a.m. after an overnight fast of 12–14 h. The laboratory kits used for the assay were supplied by Pars Azmon Inc. The assay employed an enzymatic colorimetric method with a glucose oxidase technique. The inter- and intra-assay variation coefficients for serum glucose concentration measurement are 2.2%, indicating a high level of accuracy and precision for the assay.

Exposure definition

Current and former smokers

Current smokers were defined as participants who smoked either daily or occasionally [28]. Additionally, individuals who had quit smoking for less than a year were also considered current smokers [29]. Hence, participants who had ceased smoking for over a year were categorized as former smokers. Smoking intensity was measured based on the number of cigarettes smoked per day. To adjust for differences in the intensity and duration of smoking in study participants, the pack-year index was

utilized. The pack-year was calculated by dividing the average number of cigarettes smoked per day by 20 and then multiplying it by the number of years of smoking [30].

Dietary quality index-international (DQI-I)

The dietary quality of participants was assessed using three dietary indices, including the DQI-I, Dietary Quality Index-Revised (DQI-R), and Mediterranean-Dietary Quality Index (Med.DQI). For DQI-I construction, we followed the method by Kim et al. [31]. In summary, the DQI-I is based on the North American Dietary Guidelines and emphasizes four primary elements: (1) variety in the intake of food groups including meat, poultry, fish, eggs, dairy, vegetables, fruits, grains, and beans, as well as variety within protein sources (0–20 points), (2) adequacy in the consumption of vegetables, fruits, grains, fiber, protein, and micronutrients like iron, calcium, and vitamin C (0–40 points), (3) moderation in the consumption of fat, saturated fat, cholesterol, sodium, and empty-calorie foods (0–30 points), and (4) overall balance in the ratio of macronutrients and fatty acids (0–10 points). The final DQI-I score ranges from 0 to 100, with a higher score indicating better diet quality.

Dietary quality index-revised (DQI-R)

The DQI-R is composed of ten components, four of which are identical to the original DQI (total fat, saturated fat, cholesterol, and calcium). The DQI-R now includes separate components for fruits, vegetables, grains, and iron, as well as new components for dietary moderation and diversity. Each component of the DQI-R is scored on a scale of 0 to 10, with a maximum score of 100 for the highest diet quality. Moderation in the diet pertains to the moderation of simple sugars, discretionary fat, sodium, and dietary alcohol consumption, while dietary diversity encompasses diversity in the intake of grains, fruits, vegetables, meat, and dairy products [32].

Mediterranean-dietary quality index (Med.DQI)

The Mediterranean diet has been linked to a reduced risk of chronic diseases, including all cancers, which makes the use of the Med-DQI important for assessing people's nutritional status [33]. Med-DQI consists of seven components, including meat, fish, grains, fruits and vegetables, cholesterol, saturated fatty acids, and olive oil. Each component scored 0, 1, or 2 according to the recommended guidelines and assigned individuals a score between 0 and 14, with a higher score indicating a poor-quality diet.

Outcome definition

CVDs were defined as a combined measure of coronary heart disease (CHD), stroke, or death due to cerebrovascular causes. Coronary heart disease-related events comprised cases of confirmed myocardial infarction (as determined by diagnostic electrocardiogram and biomarkers), possible myocardial infarction (established by positive electrocardiogram findings, symptoms or signs of a heart attack, and absence of biomarkers; or positive electrocardiogram findings and uncertain biomarkers), and CHD that was confirmed by angiography. The criteria for stroke were defined as a recently developed neurological impairment that persisted for a minimum of 24 h.

In the event of mortality, information was collected by an authorized local physician either from the hospital records or death certificates. The results obtained were assessed by an outcome committee that includes a chief researcher, an internist, an endocrinologist, a cardiologist, an epidemiologist, and the medical professional who compiles the outcome data.

Statistical analyses

Statistical analysis was conducted using SPSS software (Statistical Package for the Social Sciences, version 26.0, SPSS Inc., Chicago, IL, USA). The primary characteristics of the total population, consisting of current and former smokers, are presented as percentages for categorical variables and mean ± standard deviation (SD) for quantitative variables. To compare the means of quantitative and categorical variables between the two groups of current and former smokers, an independent sample t-test, and chi-square analysis were used, respectively.

We utilized a Cox proportional hazard regression to determine the hazard ratio (HR) and 95% confidence interval (CI) for smoking intensity (measured by the number of cigarettes per day), smoking duration, and intensity (measured by pack years), and DQIs with regards to incidences of both CVDs and all-cause mortality.

Heavy smokers and light smokers as well as good and poor diet quality were determined based on the median intensity and duration of smoking and the median of DQIs, respectively. We divided the population into four groups: (1) heavy smokers with poor diet quality (reference group), (2) heavy smokers with good diet quality, (3) light smokers with poor diet quality, and (4) light smokers with good diet quality.

In addition, we measured the joint association between DQIs and smoking status in four groups: (1) current smokers with poor diet quality (reference group), (2) current smokers with good diet quality, (3) former smokers with poor diet quality, and (4) former smokers with good diet quality. Model 1 was adjusted for the variables that had significant associations with CVDs in the univariate analysis, including age, systolic blood pressure, fasting serum glucose, and job status ($P<0.05$). Despite

insignificance in univariate association, due to consistency with previous studies, Model 2 additionally adjusted for body mass index, physical activity, energy intake, marriage status, and education level. For each variable, the HR, and 95% CI, were reported. The time of follow-up was computed from the date of enrollment in the study until the first occurrence of CVDs events or the last follow-up date. All *P* values were based on two-sided tests and *P* values < 0.05 were considered significant.

Results

Table 1 displays the baseline characteristics of the study population. The participants (88.60% men) had a mean age of 47.71 ± 10.61 years. The study had a mean follow-up period of 8.40 years for CVDs incidence and 8.80 years for all-cause mortality. The CVDs incidence and all-cause mortality rate in the current smoker group were 8.50% and 4.10%, respectively. Former smokers had a higher age, were more likely to be men and married, had higher fasting blood sugar and systolic blood pressure, and had a lower physical activity and employment percentage than current smokers. The incidence of CVDs in former smokers (12.60%) was higher than that in current smokers, but their overall mortality rate was similar to that of current smokers. Based on the DQI-I, DQI-R, and Med.DQI scores, the diet quality of former smokers was better than that of the current smokers group. The mean ± SD for the number of cigarettes smoked per day and pack-year index in the current smokers were equal to 8.89 ± 8.39 and 11.89 ± 12.15, respectively.

Table 2 shows that in the fully-adjusted model (model 2), those in tertile 3 of smoking intensity (HR = 2.96; 95% CI = 1.48, 5.91) and pack-year index (HR = 4.41; 95% CI = 1.61, 12.08), had a higher risk for CVDs incidence than those in tertile 1. Additionally, the highest tertile of smoking intensity (HR = 8.28; 95% CI = 2.18, 31.42) and pack-year index (HR = 4.06; 95% CI = 1.01, 16.28) were associated with a higher risk of all-cause mortality in the fully adjusted model, than the lowest tertile. There was no significant relationship between the incidence of CVDs or all-cause mortality and the DQI-I index. In the crude and adjusted models, the second tertile of the DQI-R showed a lower risk for CVDs incidence compared to the first tertile (HR for adjusted model 2 = 0.43; 95% CI: 0.19, 0.96). Furthermore, the risk of all-cause mortality was higher in the third tertile of the Med-DQI index in both adjusted models, compared to the first tertile.

The joint association between smoking and diet quality (Table 3) was assessed on two levels: smoking intensity (cigarettes per day) and smoking intensity and duration (pack-year). Concerning smoking intensity, light smokers with good diet quality in comparison with heavy smokers with poor diet quality (reference group), had a lower risk of CVDs incidence in the fully multivariate-adjusted model according to DQI-I (HR = 0.42; 95% CI: 0.18, 0.99) and DQI-R (HR = 0.35; 95% CI: 0.15, 0.83). Additionally,

Table 1 Baseline characteristics of the studied population from Tehran lipid and glucose study*

	Total population (*n* = 955)	Current smokers (*n* = 638)	Former smokers (*n* = 317)	*P*-value**
Age (year)	47.71 ± 10.61	45.65 ± 9.87	51.86 ± 10.83	**0.001**
Male (%)	88.60	86.20	93.40	**0.001**
Body mass index (kg/m^2)	27.23 ± 4.32	27.10 ± 4.49	27.49 ± 3.96	0.171
Physical activity (MET/min/wk)	2679 ± 4410	2916 ± 4810	2197 ± 3418	**0.009**
Education level (higher than diploma, %)	22.90	22.40	23.70	0.380
Job status (employed, %)	80.80	85.40	71.40	**0.001**
Marital status (married, %)	90.60	88.90	94.00	**0.011**
Fasting blood sugar (mg/dl)	99.73 ± 29.97	97.72 ± 27.62	103.78 ± 33.90	**0.006**
Systolic blood pressure (mmHg)	116.09 ± 16.52	113.97 ± 15.95	120.36 ± 16.84	**0.001**
DQI-I	63.09 ± 8.08	62.16 ± 8.12	64.97 ± 7.68	**0.001**
Variety	16.25 ± 2.97	16.22 ± 3.04	16.31 ± 2.82	0.675
Adequacy	32.08 ± 3.53	31.90 ± 3.67	32.46 ± 3.22	**0.016**
Moderation	12.33 ± 5.73	11.81 ± 5.79	13.38 ± 5.46	**0.001**
Overall balance	2.40 ± 2.50	2.22 ± 2.24	2.78 ± 2.24	**0.001**
DQI-R	70.71 ± 12.38	69.23 ± 12.49	73.67 ± 11.63	**0.001**
Dietary diversity	6.16 ± 1.32	6.15 ± 1.35	6.19 ± 1.25	0.697
Dietary moderation	6.21 ± 1.25	6.10 ± 1.29	6.42 ± 1.12	**0.001**
Med-DQI	6.05 ± 1.76	6.22 ± 1.77	5.69 ± 1.68	**0.001**
CVDs incidence (%)	9.80	8.50	12.60	**0.042**
All-cause mortality (%)	4.20	4.10	4.40	0.804

Abbreviations: DQI-I: Diet quality index-international; DQI-R: Diet quality index-revised; Med-DQI: Mediterranean-diet quality index; CVDs: Cardiovascular diseases.

* Significant differences are bolded.

***P*-values were calculated for the differences between current and former smokers using chi-square and independent sample t-test.

Table 2 The Hazard ratio and 95% CI for the incidence of CVDs and all-cause mortality based on the tertiles of diet quality indices and cigarette smoking intensity and duration among current smokers of the Tehran Lipid and Glucose Study†

	Cardiovascular diseases, HR (95%CI)			P-trend	All-cause mortality, HR (95%CI)			P-trend
	T1	T2	T3		T1	T2	T3	
Smoking intensity (cigarettes/day)								
Follow-up time (year)	8.6	8.6	7.9		9	8.8	8.6	
Person/time	1827	1982	1552		1911	2026	1666	
Incident case/Total	14/213	12/229	28/193		3/213	7/229	16/193	
Crude model	Ref	0.86 (0.39, 1.89)	**2.47 (1.27, 4.81)**	**0.001**	Ref	1.93 (0.48, 7.74)	**6.19 (1.79, 21.39)**	**0.001**
Model 1*	Ref	0.87 (0.39, 1.94)	**2.71 (1.37, 5.37)**	**0.001**	Ref	1.99 (0.47, 8.31)	**7.93 (2.19, 28.66)**	**0.001**
Model 2**	Ref	0.90 (0.40, 2.02)	**2.96 (1.48, 5.91)**	**0.001**	Ref	2.10 (0.49, 9.05)	**8.28 (2.18, 31.42)**	**0.001**
Smoking intensity and duration (Pack. year)								
Follow-up time (year)	8.8	8.5	7.9		8.9	8.9	8.6	
Person/time	1616	1597	1449		1644	1663	1560	
Incident case/Total	6/183	12/186	29/180		3/183	5/186	17/180	
Crude model	Ref	2.37 (0.83, 6.75)	**6.26 (2.41, 16.28)**	**0.001**	Ref	1.29 (0.29, 5.79)	**5.93 (1.73, 20.37)**	**0.001**
Model 1	Ref	1.80 (0.62, 5.24)	**4.03 (1.49, 10.87)**	**0.001**	Ref	0.98 (0.20, 4.62)	**4.43 (1.15, 17.03)**	**0.002**
Model 2**	Ref	1.82 (0.62, 5.27)	**4.41 (1.61, 12.08)**	**0.001**	Ref	0.96 (0.20, 4.65)	**4.06 (1.01, 16.28)**	**0.007**
DQI-I								
Follow-up time (year)	8.4	8.5	8.2		8.8	8.8	8.8	
Person/time	1808	1816	1761		1878	1875	1875	
Incident case/Total	14/213	18/213	22/212		7/213	11/213	8/212	
Crude model	Ref	1.23 (0.61, 2.47)	1.32 (0.66, 2.63)	0.441	Ref	1.36 (0.52, 3.59)	0.95 (0.33, 2.72)	0.897
Model 1*	Ref	1.16 (0.57, 2.38)	1.23 (0.61, 2.48)	0.555	Ref	1.16 (0.43, 3.15)	0.78 (0.27, 2.25)	0.607
Model 2**	Ref	1.13 (0.55, 2.32)	1.15 (0.56, 2.36)	0.711	Ref	1.22 (0.44, 3.38)	0.90 (0.30, 2.68)	0.814
DQI-R								
Follow-up time (year)	8.5	8.6	8.00		9	8.8	8.5	
Person/time	2308	1713	1364		2419	1756	1453	
Incident case/Total	26/268	10/200	18/170		12/268	8/200	6/170	
Crude model	Ref	**0.45 (0.21, 0.96)**	0.94 (0.50, 1.76)	0.497	Ref	0.78 (0.31, 1.99)	0.63 (0.22, 1.81)	0.379
Model 1*	Ref	**0.45 (0.20, 0.98)**	0.95 (0.51, 1.78)	0.542	Ref	0.59 (0.22, 1.56)	0.56 (0.19, 1.62)	0.223
Model 2**	Ref	**0.43 (0.19, 0.96)**	0.84 (0.43, 1.66)	0.359	Ref	0.55 (0.20, 1.52)	0.54 (0.17, 1.67)	0.220
Med.DQI								
Follow-up time (year)	8.1	8.4	8.7		8.6	8.9	8.9	
Person/time	1830	2253	1302		1945	2353	1330	
Incident case/Total	23/226	24/264	7/148		7/226	11/264	8/148	
Crude model	Ref	0.93 (0.51, 1.68)	0.51 (0.21, 1.21)	0.127	Ref	1.09 (0.40, 2.95)	1.81 (0.65, 4.49)	0.222

Table 2 (continued)

	Cardiovascular diseases, HR (95%CI)			P-trend	All-cause mortality, HR (95%CI)			P-trend
	T1	T2	T3		T1	T2	T3	
Model 1*	Ref	1.08 (0.59, 1.97)	0.62 (0.26, 1.47)	0.289	Ref	1.59 (0.58, 4.37)	**3.42 (1.15, 10.21)**	**0.023**
Model 2**	Ref	1.09 (0.59, 2.04)	0.62 (0.26, 1.50)	0.300	Ref	1.56 (0.55, 4.43)	**3.45 (1.12, 10.57)**	**0.025**

Abbreviations: DQI-I: Diet quality index-international; DQI-R: Diet quality index-revised; Med-DQI: Mediterranean-diet quality index.

*Adjusted for age, systolic blood pressure, fasting blood sugar, and job status.

**Additionally, adjusted for body mass index, physical activity, calorie intake, marriage status, and education level.

† Significant relations are bolded.

only light smokers with good diet quality showed a lower risk for all-cause mortality compared to the reference group based on DQI-I (HR=0.20; 95% CI: 0.05, 0.77) and DQI-R (HR=0.19; 95% CI: 0.03, 0.98) in the fully multivariable-adjusted model. In addition, there was no significant joint association between Med-DQI and smoking intensity with CVDs incidence and all-cause mortality. Concerning the pack-year index, light smokers with a good DQI-R had a lower risk of CVDs than heavy smokers with poor diet quality in crude and adjusted models. (HR for adjusted model 2=0.32; 95% CI: 0.12, 0.83) Additionally, light smokers with good diet quality had a lower risk of all-cause mortality in crude and adjusted models according to the DQI-I (HR for adjusted model 2=0.08; 95% CI: 0.01, 0.69) and Med.DQI (HR for adjusted model 2=0.07; 95% CI: 0.00, 0.81).

In Supplementary Table 1, the risk of CVDs incidence and all-cause mortality was reported in former smokers compared to current smokers. After controlling for the confounding effect of various variables, former smokers had a 61% lower risk of all-cause mortality compared to current smokers (HR=0.39; 95% CI: 0.17, 0.90). The risk of CVDs incidence and all-cause mortality among former smokers was not significantly different from that of light smokers, but in a fully adjusted model, former smokers had a lower risk of CVDs incidence (HR=0.53; 95% CI: 0.31, 0.89) and mortality (HR=0.22; 95% CI: 0.08, 0.55) when compared to heavy smokers.

Figure 2 and Supplementary Table 2, indicate the joint association between smoking and diet quality indices among current and former smokers. Former smokers with poor diet quality had a lower risk of all-cause mortality in adjusted models based on the DQI-I (HR=0.05; 95% CI: 0.00, 0.52) and DQI-R (HR=0.22; 95% CI: 0.05, 0.92) than current smokers with poor diet quality. According to the Med-DQI, former smokers who adhere to good diet quality, compared to current smokers with poor diet quality, had a lower risk for all-cause mortality in Model 2 (HR=0.10; 95% CI: 0.02, 0.45). In addition, current smokers with good diet quality show a lower risk of mortality compared to current smokers with poor diet quality after accounting for potential covariates in the fully adjusted model (HR=0.26; 95% CI: 0.08, 0.80).

Discussion

We observed that cigarette smoking intensity and duration are independent contributors to CVDs incidence and all-cause mortality. The lower diet quality of current smokers, according to the Med-DQI, can increase the risk of mortality up to threefold. Light smokers with good diet quality had a lower risk of CVDs incidence and all-cause mortality compared to heavy smokers with poor diet quality. Current smokers with good Med-DQI had a lower risk of all-cause mortality compared to those with

Table 3 The Hazard ratio and 95% CI for the incidence of CVDs and mortality based on the diet quality indices joint association with smoking intensity and duration†

	Hazard Ratio (95%CI)			
	Heavy smoker-poor diet quality	**Heavy smoker-good diet quality**	**Light smoker-poor diet quality**	**Light smoker-good diet quality**
Smoking intensity				
CVDs				
DQI-I				
Crude model	Ref	1.34 (0.67, 2.65)	0.56 (0.25, 1.28)	0.47 (0.20, 1.06)
Model 1*	Ref	1.39 (0.70, 2.77)	0.62 (0.27, 1.41)	0.46 (0.20, 1.07)
Model 2**	Ref	1.32 (0.66, 2.66)	0.62 (0.27, 1.41)	**0.42 (0.18, 0.99)**
DQI-R				
Crude model	Ref	1.08 (0.53, 2.17)	0.52 (0.24, 1.11)	**0.40 (0.17, 0.92)**
Model 1*	Ref	1.05 (0.52, 2.14)	0.53 (0.25, 1.15)	**0.39 (0.17, 0.90)**
Model 2**	Ref	0.99 (0.48, 2.05)	0.53 (0.24, 1.15)	**0.35 (0.15, 0.83)**
MED-DQI				
Crude model	Ref	2.03 (0.97, 4.28)	0.68 (0.26, 1.79)	0.70 (0.29, 1.65)
Model 1*	Ref	1.91 (0.90, 4.06)	0.92 (0.34, 2.45)	0.58 (0.24, 1.40)
Model 2**	Ref	1.87 (0.88, 3.99)	0.91 (0.34, 2.43)	0.56 (0.23, 1.35)
All-cause mortality				
DQI-I				
Crude model	Ref	0.74 (0.27, 2.06)	0.50 (0.17, 1.47)	**0.25 (0.06, 0.91)**
Model 1*	Ref	0.62 (0.22, 1.75)	0.48 (0.16, 1.43)	**0.19 (0.05, 0.74)**
Model 2**	Ref	0.68 (0.24, 1.93)	0.49 (0.16, 1.50)	**0.20 (0.05, 0.77)**
DQI-R				
Crude model	Ref	1.67 (0.62, 4.46)	0.78 (0.27, 2.26)	0.25 (0.05, 1.20)
Model 1*	Ref	1.60 (0.58, 4.43)	0.83 (0.28, 2.43)	0.21 (0.04, 1.02)
Model 2**	Ref	1.62 (0.58, 4.52)	0.85 (0.28, 2.54)	**0.19 (0.03, 0.98)**
MED-DQI				
Crude model	Ref	1.89 (0.65, 5.45)	0.77 (0.20, 2.89)	0.50 (0.13, 1.87)
Model 1*	Ref	1.31 (0.44, 3.92)	0.96 (0.25, 3.64)	0.30 (0.07, 1.18)
Model 2**	Ref	1.40 (0.46, 4.24)	1.03 (0.27, 3.91)	0.29 (0.07, 1.21)
Smoking duration and intensity				
Cardiovascular diseases				
DQI-I				
Crude model	Ref	0.93 (0.46, 1.87)	**0.29 (0.11, 0.80)**	**0.35 (0.14, 0.85)**
Model 1*	Ref	0.94 (0.46, 1.90)	0.46 (0.17, 1.27)	0.46 (0.19, 1.11)
Model 2**	Ref	0.90 (0.44, 1.84)	0.44 (0.16, 1.23)	0.41 (0.16, 1.03)
DQI-R				
Crude model	Ref	0.71 (0.34, 1.47)	**0.28 (0.11, 0.70)**	**0.31 (0.12, 0.77)**
Model 1*	Ref	0.45 (0.18, 1.15)	0.45 (0.18, 1.15)	**0.38 (0.15, 0.96)**
Model 2**	Ref	0.64 (0.30, 1.35)	0.43 (0.17, 1.10)	**0.32 (0.12, 0.83)**
MED-DQI				
Crude model	Ref	1.36 (0.66, 2.79)	**0.24 (0.07, 0.87)**	0.52 (0.22, 1.23)
Model 1*	Ref	1.27 (0.62, 2.62)	0.44 (0.12, 1.61)	0.59 (0.24, 1.42)
Model 2**	Ref	1.25 (0.60, 2.59)	0.43 (0.11, 1.56)	0.55 (0.22, 1.37)
All-cause mortality				
DQI-I				
Crude model	Ref	0.70 (0.27, 1.79)	**0.27 (0.07, 0.96)**	**0.07 (0.01, 0.59)**
Model 1*	Ref	0.61 (0.23, 1.57)	0.41 (0.11, 1.53)	**0.08 (0.01, 0.65)**
Model 2**	Ref	0.66 (0.25, 1.71)	0.44 (0.11, 1.75)	**0.08 (0.01, 0.69)**
DQI-R				
Crude model	Ref	1.36 (0.56, 3.28)	0.42 (0.13, 1.35)	**0.11 (0.01, 0.89)**
Model 1*	Ref	1.32 (0.54, 3.22)	0.75 (0.22, 2.51)	0.12 (0.01, 1.01)
Model 2**	Ref	1.35 (0.55, 3.34)	0.84 (0.24, 2.92)	0.11 (0.01, 1.02)

Table 3 (continued)

	Hazard Ratio (95%CI)			
	Heavy smoker-poor diet quality	**Heavy smoker-good diet quality**	**Light smoker-poor diet quality**	**Light smoker-good diet quality**
MED-DQI				
Crude model	Ref	1.75 (0.66, 4.61)	0.50 (0.12, 2.00)	**0.11 (0.01, 0.95)**
Model 1*	Ref	1.40 (0.52, 3.75)	1.10 (0.26, 4.61)	**0.08 (0.01, 0.81)**
Model 2**	Ref	1.42 (0.52, 3.90)	1.24 (0.29, 5.22)	**0.07 (0.00, 0.81)**

Abbreviations: DQI-I: Diet quality index-international; DQI-R: Diet quality index-revised; Med-DQI: Mediterranean-diet quality index

*Adjusted for age, systolic blood pressure, fasting blood sugar, and job status.

**Additionally, adjusted for body mass index, physical activity, calorie intake, marriage status, and education level.

† Significant HRs are bolded.

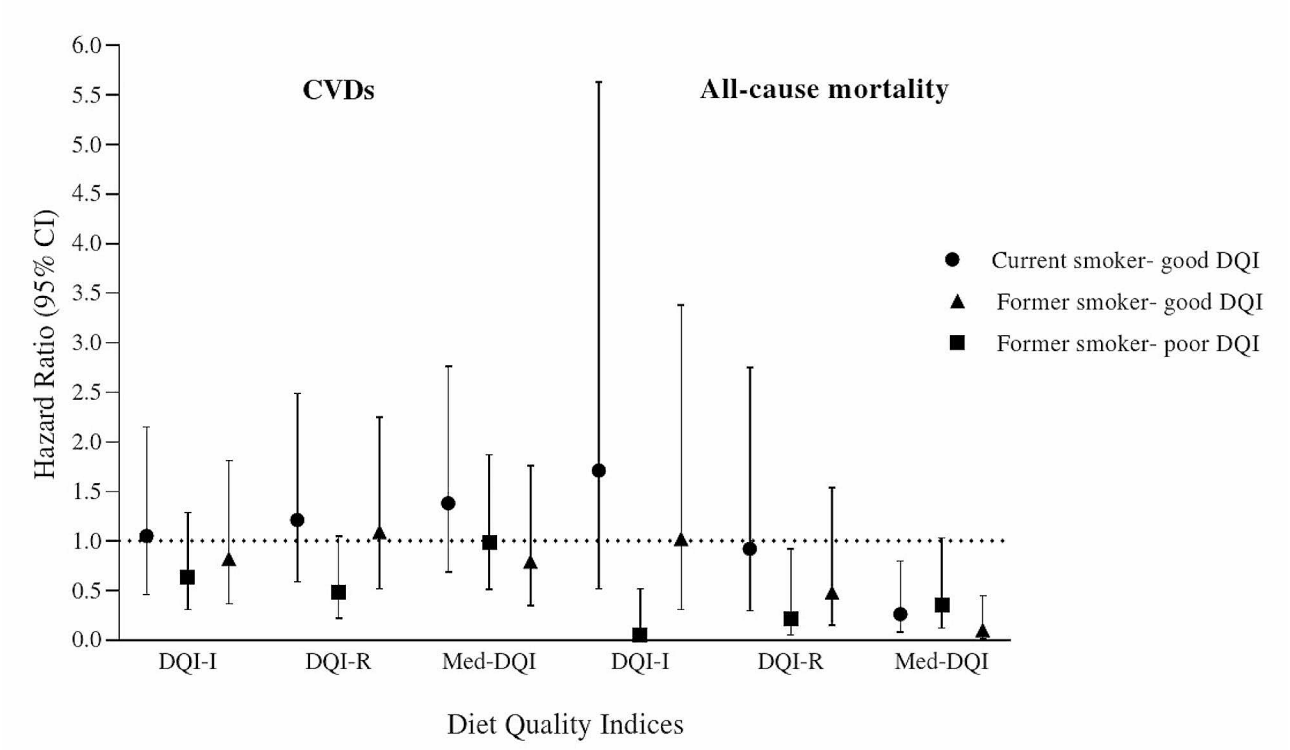

Fig. 2 The Hazard ratio and 95% CI for incidence of CVDs and all-cause mortality based on the diet quality indices joint association with smoking status (Current smoker-poor DQI considered as the reference group)

poor diet quality. This lower risk was more pronounced for former smokers than for current smokers.

Our study's findings align with previous research [34, 35], indicating that cigarette smoking is strongly linked with CVDs incidence and all-cause mortality. Cigarette smoke contains polyaromatic hydrocarbons and oxidant gases [36]. It can impact CVDs by affecting all stages of atherosclerosis, vascular function, insulin sensitivity, serum lipid profile, advanced glycation end products (AGEs) synthesis, and gene methylation [35, 37, 38]. Also, according to our study, a low-quality diet among current smokers is linked to a higher risk of all-cause mortality. Studies that have measured the dietary quality of smokers using the DQI are scarce, but some studies have measured diet quality in the general population. The results from the Korean National Health and Nutrition Examination Surveys indicated that individuals with CVDs tend to have poorer diet quality than those without CVDs, as measured by DQI-I [39]. Asghari et al., have found a direct association between higher DQI-I scores and serum HDL-C levels [40]. However, a cross-sectional study by Daneshzad et al. did not find any significant association between DQI-I score and CVDs risk factors in type 2 diabetic women, possibly due to low statistical power [19].

According to our findings light smokers with a good diet quality had better outcomes than heavy smokers with a poor diet quality. To the best of our knowledge, no study has examined the joint association between smoking intensity, duration, and diet quality and the incidence of CVDs and all-cause mortality. Nonetheless, some studies examined the relationship between certain dietary constituents with clinical outcomes among smokers. According to Clark et al., dietary fiber intake can reduce the negative effects of second-hand smoke on mortality caused by coronary artery diseases [12]. Geng et al. also observed that individuals who are genetically predisposed to smoking and have lower DASH (dietary approach to stop hypertension) scores are at a higher risk of CVDs mortality [4]. Additionally, our study findings indicate that current smokers who maintain good diet quality have a lower risk of all-cause mortality as compared to current smokers with poor diet quality. Dauchet and colleagues have suggested that male smokers who consume more fruits and vegetables may have a lower risk of CVDs [14]. A review conducted by Vardavas et al. indicated that the health effects of smoking, including CVDs, and adherence to the Mediterranean diet, as a high-quality diet, may partially interact over time [38].

In general, smokers are more likely to experience negative clinical outcomes, which may be partially attributable to their poor nutritional status [9, 41]. Studies suggest that smokers are more susceptible to oxidative damage as a result of insufficient nutrient intake and the impact of smoking on nutrient metabolism [42]. Furthermore, smokers are more likely to consume excessive amounts of sodium [43], suffer from a decreased sense of taste [10], and suppressed appetite [44]. Smokers often tend to opt for less nutritious food options and consume less fiber, carotene, vitamin C, iron, and polyunsaturated fatty acids (PUFA) [42]. Notably, smoking can significantly affect the interaction between omega-3 and omega-6 PUFAs, and alter their metabolism [38]. Smokers have been shown to have lower adherence to high-quality diets like the Mediterranean diet [45]. Additionally, adherence to the Mediterranean diet has been linked to smoking cessation [46]. A study conducted by Roswall et al. found that adhering to a healthy Nordic food index had beneficial effects on CVDs risk only among former smokers [47]. They suggest that smoking cessation is associated with dietary changes that promote a healthier lifestyle, including an increase in fruit and vegetable consumption [47].

Several mechanisms are responsible for the observed joint association between smoking and diet quality. It has been observed that smokers had a lower level of antioxidants in their blood circulation [14]. However, the intake of fruits and vegetables with a high content of antioxidants was effective in controlling smoking-related oxidative damage [14]. Furthermore, Smoking's negative effects on blood viscosity can be mitigated by flavonoids, which have been shown to inhibit platelet aggregation [14]. The fruit and vegetable intake has been linked to lower levels of C-reactive protein. Lower concentrations of C-reactive protein may reduce the negative effects of smoking on atherosclerosis and related mortality [14, 48]. In addition, a high-quality diet like the DASH diet has been shown to reduce oxidative stress and endothelial dysfunction through the lowering of dietary acid load [4]. Furthermore, adhering to a healthy diet may mitigate the deleterious effects of smoking on CVDs by promoting healthy gut microbiota [4]. The relationship between diet and smoking-associated mortality indicates that diet may serve as an indirect preventative measure by mitigating risk factors such as obesity and type 2 diabetes [4]. The Mediterranean diet, in particular, offers numerous health benefits like maintaining the ideal omega-6/omega-3 fatty acid ratio, modifying cell membrane composition and function, and gene expression [38]. Additionally, the Mediterranean diet can lower circulating low and very low-density lipoprotein cholesterol (LDL-C and VLDL-C) levels while increasing beneficial HDL-C [38].

However, we did not observe a significantly lower risk for CVDs incidence among heavy smokers with good diet quality compared to heavy smokers with poor diet quality. Millen et al. reported that smokers who adopt a heart-healthy diet are still at a higher risk of CVDs and all-cause mortality compared to non-smokers who follow a heart-healthy diet [3]. Studies have found that smoking-generated free radicals can counteract the dietary antioxidant effect due to oxidation and exert a pro-oxidative effect [49]. Also, we did not find a significant decrease in the risk of CVDs among former smokers, regardless of their diet quality, compared to current smokers with poor diet quality. Smoking can affect the hypothalamus, which reduces appetite and increases the level of catecholamines. This results in weight loss due to increased peripheral tissue energy consumption. However, after quitting smoking, with the elimination of nicotine's appetite suppression effect, individuals may consume more food and experience weight gain. This weight gain, combined with other unknown confounding variables, could be the reason for the lack of statistical significance [44].

This study has several strengths, such as its novelty in the topic, prolonged follow-up period, focused research on the combined impact of smoking and diet quality, use of validated and reproducible FFQ to collect dietary data, and control for various potentially confounding variables. However, this study has some limitations that need to be considered. First, the number of participants in the pack-year group decreased due to incomplete data related to smoking duration. Second, the smaller sample size in the pack-year group resulted in lower statistical power. Third, like all observational studies, this study may be prone

to measurement bias. Fourth, the study did not confirm the level of smoking intensity using serum markers such as cotinine. Additionally, no information was provided about changes in smoking habits during the study intervals. Fifth, the DQI-R computation did not include alcohol consumption because of a lack of available data. Lastly, because this was an observational study, unknown residual confounding effects could not be eliminated.

Conclusions

In conclusion, the results of this prospective cohort study enhance the existing knowledge that smoking intensity, duration, and poor diet quality are significant risk factors for the incidence of CVDs and all-cause mortality. Light and former smokers exhibited a lower risk of developing CVDs and mortality, while a high-quality diet further strengthened this protective effect. Although smoking cessation remains the optimal approach to avoiding the negative health consequences of smoking, adherence to a high-quality diet could confer additional support, which could have substantial implications for clinical outcomes and public health.

Abbreviations

AGEs	Advanced Glycation End Products
CHD	Coronary Heart Disease
CI	Confidence Interval
CVDs	Cardiovascular Diseases
DALYs	Disability-Adjusted Life Years
DASH	Dietary Approaches to Stop Hypertension
DQI-I	Diet Quality Index-International
DQI-R	Diet Quality Index-Revised
DQI	Diet Quality Index
FCT	Food Composition Table
FFQ	Food Frequency Questionnaire
HDL-C	High-density Lipoprotein Cholesterol
HR	Hazard Ratio
LDL-C	Low-density Lipoprotein Cholesterol
MAQ	Modifiable Activity Questionnaire
Med-DQI	Mediterranean-Diet Quality Index
MENA	Middle East and North Africa
MET-min/week	Metabolic Equivalent minutes per week
PUFA	Polyunsaturated Fatty Acid
SD	Standard Deviation
TLGS	Tehran Lipid and Glucose Study
USDA	United States Department of Agriculture
VLDL-C	Very Low-density Lipoprotein Cholesterol

Supplementary Information

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Supplementary Material 1

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None.

Author contributions

MN and STR conceived the study. MN and FT carried out the analysis and wrote the manuscript. HF and NM carried out independent manuscript revision and language editing. PM and FA, providing supervision throughout the article writing process. The manuscript has been read and approved by all authors.

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Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate

The study followed the Declaration of Helsinki by obtaining informed consent from participants and receiving approval from the Ethics Committee of the Iran University of Medical Sciences (IR.IUMS.REC.1402.138).

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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