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Adherence to the Healthy Nordic Food Index and the incidence of acute myocardial infarction and mortality among patients with stable angina pectoris

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Abstract

Background: The Healthy Nordic Food Index (HNFI) has been associated with beneficial effects on markers of cardiovascular disease (CVD). Whether such effects are present among patients with established coronary heart disease is unknown. In the present study, we investigated the association between adherence to the HNFI and the risk of acute myocardial infarction (AMI) (fatal or nonfatal) and death among patients with stable angina pectoris.

Methods: In the Western Norway B-vitamin Intervention Trial, participants completed a 169-item semi-quantitative food frequency questionnaire. The HNFI was calculated from six food groups (fish, cabbage, apples/pears, root vegetables, whole grain bread and oatmeal), scoring 0–6. Three adherence groups were defined: 0–1 points (low), 2–3 points (medium) or 4–6 points (high). Cox regression analyses investigated associations between adherence to the HNFI and outcomes.

Results: Among 2019 men (79.7%) and women with mean age of 61.7 years, 307 patients experienced an AMI event during a median (25th and 75th percentiles) follow-up of 7.5 (6.3 and 8.7) years. Median follow-up for total mortality was 10.5 (9.3 and 11.7) years; 171 patients died from CVD and 380 from any cause. No association between HNFI and the risk of AMI was detected. However, the HNFI was associated with a reduced risk of all-cause death, both by linear estimates [hazard ratio (95% confidence interval = 0.91 (0.84–0.98)] and by comparison of the highest with the lowest adherence group [hazard ratio (95% confidence interval = 0.70 (0.52–0.95)].

Conclusions: The results of the present study suggest that a Healthy Nordic diet may reduce mortality in patients with established CVD.

Introduction

Cardiovascular disease (CVD) is the number one cause of death globally, and 80% of all CVD deaths are a result of acute myocardial infarction (AMI) or stroke [(1)]. Despite optimised medical treatment, patients suffering from coronary heart disease and stable angina have an increased risk of new cardiovascular events and mortality [(2)]. There is no

doubt that diet influences the development and progression of CVD. Dietary intakes rich in fish [3], vegetables [4] or whole grain [5] have been shown to be inversely associated with CVD development and progression. Because food intakes usually show high degrees of collinearity [6], dietary pattern analysis has been developed to evaluate the effect of the whole diet on disease development and progression [7,8].

Dietary pattern analysis can follow a hypothesis-free data analysis [9] or follow the concept that a predefined dietary pattern has a particular effect on disease [10–12]. In particular, the so-called Mediterranean Diet has been described and investigated in relation to CVD. Adherence to the diet is measured using a scoring system, where a score (zero or one) is given for a certain amount of consumption of each food component. Sex-specific median intakes are used as a cut-off. The score then reflects adherence to the diet and can be analysed with respect to biomarkers or risk factors, and clinical outcomes [13].

The use of the Mediterranean Diet score may not be suitable for populations in areas outside southern Europe because the score would not reflect local dietary habits and would thus cause misclassification [14]. Thus, other local food scores have been defined, especially in the Nordic countries, which have a number of specific dietary traditions in common [15]. Among them, the Nordic diet has been characterised [15,16] and analysed with respect to biomarkers [17] and cardiovascular risk factors in healthy and hypercholesterolemic populations [18,19]. The Nordic Diet is probably more acceptable for populations in the Nordic countries and might be easier to adhere to than the Mediterranean Diet [20,21].

However, there are different definitions of the Nordic diet, depending on geographical location and focus of the study [15]. In the present study, we use the approach of the Healthy Nordic Food Index (HNFI) based on scores for intake of fish, cabbage, fruits and berries, root vegetables, whole grain or rye bread and oatmeal [14]. It has been shown that high adherence to the score is associated with a healthier lifestyle [22] , as well as a lower risk of myocardial infarction [23] and total mortality [14], in healthy subjects. This led to the hypothesis that a high score of the HNFI is protective of future AMI and cardiovascular and total mortality among patients at high risk. Accordingly, we analysed whether adherence to the HNFI was associated with the risk of AMI and mortality in a cohort of Norwegian patients with stable angina pectoris.

Materials and methods

Study population

From 1999 to 2004, 3090 adult patients at two Norwegian University Hospitals in Bergen and Stavanger undergoing diagnostic coronary angiography because of suspected coronary artery disease (CAD) or aortic stenosis were recruited to the Western Norway B-Vitamin Intervention Trial (WENBIT). The main aim was to investigate clinical outcomes of homocysteine-lowering B-vitamin treatment [24,25]. No significant effect of the B-vitamin intervention was found on cardiovascular outcomes or mortality [25,26]. Inclusion criteria for randomisation were men and women aged 18 years or older undergoing coronary angiography for suspected CAD and/or aortic valve stenosis at two University hospitals in Western Norway. Exclusion criteria were unavailability for follow-up, participation in other trials, known alcohol abuse, serious mental illness, cancer disease or patients not filling out the food frequency questionnaire (FFQ) [26]**.** Information on dietary habits was collected by a self-administered FFQ from 2484 participants. After further exclusion of participants who had more than one blank page on the FFQ (n = 96) and participants who had very low [<30 MJ (<717 kcal) for women and <33 MJ (<789 kcal) for men] or very high [<150 MJ (>3585 kcal) for women and <175 MJ (>4182 kcal) for men] estimated daily energy intakes (n = 37), FFQ data from 2351 participants were considered to have satisfactory quality. Patients with a history of acute coronary syndrome were excluded because of a tendency to change their diet [27]. Thus, data from 2019 patients with stable angina patients were used for the current analysis (Fig. 1). All participants provided their written consent. The study protocol was in accordance with the principles of the *Declaration of Helsinki* and approved by the Data Inspectorate and the Regional Committee for Medical and Health Research Ethics, and the Norwegian Medicines Agency.

Food frequency questionnaire

The FFQ was handed out after coronary angiography during inclusion in WENBIT, filled in at home and returned to the study centre either at the follow-up visit scheduled 1 month later or by mail ahead of this appointment. The 10-page questionnaire included 169 food items grouped according to Norwegian meal patterns and was designed to obtain information on habitual food intake during the past year. The frequency of consumption was given per day, week or month, depending on the items in question. Portion sizes were given as household measures or units such as slices, pieces, spoons or glasses. Average daily intake was used in the analyses. The FFQ was developed at the Department of Nutrition at the University of Oslo (Oslo, Norway) and has been validated [28–31]. Estimations of nutrient and food intakes were performed using a food database and a bespoke software system (KOSTBEREGNINGSSYSTEM, version 3.2; Department of Nutrition,

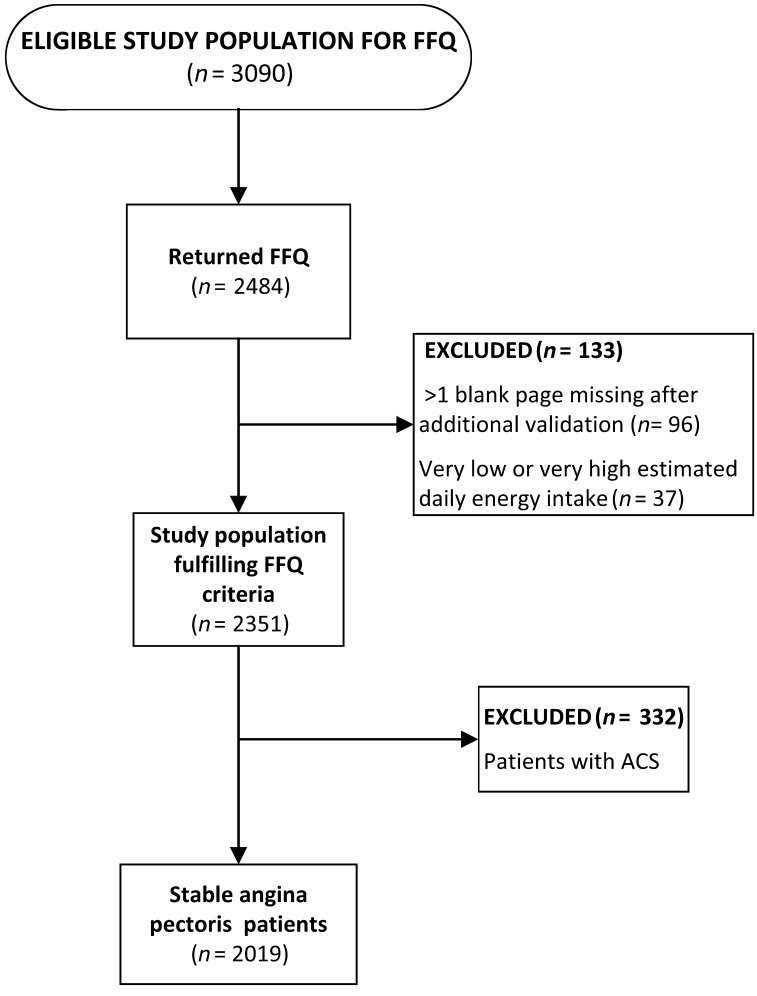

Figure 1 Flow of included and excluded patients to the current study cohort. ACS, acute coronary syndrome; FFQ, food frequency questionnaire.

University of Oslo, Oslo, Norway). This database is based on the official Norwegian Food Table [30].

The Healthy Nordic Food Index

The HNFI includes dietary items likely to have beneficial health effects when the consumption is high [14] and is based on six typical food groups consumed in the Nordic countries (fish, cabbage, apple and pears, root vegetables, rye bread and oatmeal). Similar to a previous study [32], we replaced rye bread with wholegrain bread because information on rye bread specifically was not included in the FFQ.

The sex-specific medians for consumption of each food group were calculated (see Supporting information, Table S1). Participants with a dietary intake equal to or above the median intake were given one point for each of the six food groups, and zero points were given for intakes below the median. The median intake for oatmeal was zero g day^{-1} for both women and men because more than 50% of the cohort did not consume oatmeal. Instead, participants with any intake received one point. Accordingly, one point was assigned for the following reported daily intakes for men: fish ≥103 g, cabbages ≥27 g, apples and pears ≥38 g, root vegetables ≥48 g, oatmeal ≥1 g, whole grain bread ≥2 slices (80 g). For women, one point was given for daily intakes: fish ≥78 g, cabbages ≥27 g, apples and pears ≥38 g, root vegetables ≥50 g, oatmeal ≥1 g, whole grain bread ≥3 slices (120 g).

The total score was summed up for each participant who could achieve a maximum score of six points (see Supporting information, Table S2). To ensure a sufficient number of cases within each exposure category, the sex-specific scores were further categorised into three adherence groups: low (0–1 points), medium (2–3 points) and high (4–6 points) adherence.

Laboratory analyses and assessment of other covariates

Demographic, clinical and routine laboratory data were obtained by study personnel at the two study centres, as

described previously [33]. Standard blood laboratory parameters were analysed from fresh samples in accordance with routine protocols at the hospital laboratories and the analytical information on measurements of apolipoprotein A1, apolipoprotein B, cotinine and low-density lipoprotein cholesterol is described in detail elsewhere [27].

Diabetes mellitus was classified in accordance with the existing diagnosis (prevalent diabetes either yes or no) or based on baseline fasting serum glucose ≥7.0 mmol L^{-1} or nonfasting glucose ≥11.1 mmol L^{-1} [34]. Smokers include self-reported current smokers, those reported having quit within the last 4 weeks and patients with plasma cotinine ≥85 nmol L^{-1} at baseline [35]. Left ventricular ejection fraction was determined by ventriculography or echocardiography; values <50% were classified as impaired systolic function. The extent of CAD was angiographically graded as nonsignificant stenosis (luminal narrowing <50%), or as having single-, double- or triple-vessel disease [36].

Endpoints and follow-up

The study endpoints were fatal and nonfatal AMI, cardiovascular and all-cause death. Patients were followed until 1 January 2010 for AMI events through the Cardiovascular Disease in Norway project (CVDNOR www.cvdnor.no) [37,38] and until 1 January 2013 for cardiovascular and total death through the Norwegian Cause of Death Registry. The incidence of cardiovascular death included deaths coded as I00 to I96 or R99 according to the International Classification of Disease, 10th Revsison [39].

Statistical analysis

Baseline and dietary variables are reported as the mean (SD), medians (25th and 75th percentiles) or proportions as appropriate. Dietary intakes of macronutrients are presented as proportions of total energy intake, whereas intakes of food items are given as densities (g 1000 $kcal^{-1}$). All participants were ranked according to the three HNFI adherence groups (0–1, 2–3 and 4–6 points). An association between the HNFI adherence groups and baseline variables was assessed using linear regression for continuous variables and logistic regression or chi-squared for categorical variables (Table 1).

Associations between adherence to the HNFI and subsequent risk of endpoints during follow-up were evaluated by Cox proportional hazard models. Risk associations were explored by comparing the two highest HNFI groups with the lowest adherence group (0–1 points) and by linear trends across these groups, as well as according to the HNFI score. Hazard ratios (HRs) and 95% confidence intervals (CI) are reported. For the endpoint AMI, analyses were repeated for fatal and nonfatal AMI, separately.

Three different models are presented for each outcome. A basic model was controlled for age and sex (Model 1). Additionally, we adjusted for energy intake (Model 2) to reduce confounding by differences in energy intake. In Model 3, we further adjusted for alcohol intake (g/day/1000kcal) and clinically relevant covariates: diabetes mellitus (yes/no); hypertension (yes/no); physical activity (never; 1 day $week^{-1}$; 2–3 times $week^{-1}$; at least 4 days $week^{-1}$); current smoking (yes/no); and use of statins at discharge form hospital (yes/no). Additional adjustment for waist circumference, acetylsalicylic acid, angiotensin-converting enzyme inhibitors and/or angiotensin receptor blockers, β-blockers, loop diuretics, left ventricle ejection fraction, plasma concentration of cholesterol, and a history of percutaneous coronary intervention did not affect the estimates (data not shown); thus, these factors were not included in the final analyses.

Kaplan–Meier survival plots were applied to illustrate the survival time for sex-specific outcomes. Statistical analyses were performed using SPSS, version 24 (IBM Corp., Armonk, NY, USA). $P < 0.05$ was considered statistically significant.

Results

Baseline characteristics

Characteristics are presented across the three HNFI adherence groups (Table 1). Overall, 19.4% of patients were classified as having low adherence (0–1 points), 44.7% as having medium adherence (2–3 points) and 35.9% as having high adherence (4–6 points). The age of subjects at inclusion ranged from 28 to 85 years, with a mean (SD) age of 61.7 (10) years.

Participants who scored high on the HNFI were older ($P = 0.001$), more likely to be men ($P = 0.02$), physically active ($P = 0.002$) and to have a lower serum total cholesterol ($P < 0.001$), low-density lipoprotein cholesterol ($P < 0.001$), apolipoprotein B ($P < 0.001$) and triglycerides ($P = 0.01$). These patients were also less likely to be current smokers ($P < 0.001$), to have a history of percutaneous coronary intervention ($P = 0.002$) and to have a reduced ejection fraction <50% ($P = 0.01$). Acetylsalicylic acid (89%), statins (88%) and β-blockers (77%) were used by a majority of the participants after baseline coronary angiography. Participants with high HNFI adherence were less likely to use loop diuretics ($P = 0.01$).

High adherence to the HNFI corresponded to higher intakes of total energy, higher energy percentage (E%) from proteins and carbohydrates ($P < 0.001$ for all). As

Table 1 Baseline characteristics of patients with stable angina pectoris

Characteristics	Total	Healthy Nordic Food Index groups			*P*-trend[†]
		0–1 (lowest)	2–3	4–6 (highest)	
Patients, *n* (%)	2019	392 (19.4)	903 (44.7)	724 (35.9)	
Age (years)	61.8 (9.7)*	60.1 (10.3)	62.0 (10.1)	62.4 (8.8)	0.001
Male sex, *n* (%)	1610 (79.7)	309 (78.8)	698 (77.3)	603 (83.3)	0.03
Body mass index (kg m^{-2})	26.4 (3.7)*	26.4 (4.1)	26.3 (3.7)	26.4 (3.7)	0.62
Waist circumference (cm)	96.4 (11)*	96.3 (11.9)	96.1 (10.5)	96.9 (11.4)	0.27
Cardiovascular history, *n* (%)					
Percutaneous coronary intervention	452 (22.4)	108 (27.6)	204 (22.6)	140 (19.3)	0.002
Coronary artery bypass graft surgery	290 (14.4)	49 (12.5)	126 (14.0)	115 (15.9)	0.11
Acute myocardial infarction	867 (42.9)	190 (48.5)	373 (41.3)	304 (42.0)	0.08
Coronary risk factors, *n* (%)					
Hypertension	958 (47.4)	167 (42.6)	441 (48.8)	350 (48.3)	0.060
Diabetes mellitus[‡]	622 (30.8)	111 (28.3)	289 (32.0)	222 (30.7)	0.29
Current smoker[§]	593 (29.4)	159 (40.6)	265 (29.3)	169 (23.3)	<0.001
Physical activity[¶]:					
Never	109 (5.4)	29 (7.4)	52 (5.8)	28 (3.9)	0.002
1 day $week^{-1}$	375 (18.6)	95 (24.2)	169 (18.7)	11 (15.3)	
2–3 times $week^{-1}$	641 (31.9)	105 (26.8)	281 (31.1)	255 (35.2)	
At least 4 days $week^{-1}$	462 (23.0)	80 (20.4)	205 (22.7)	177 (24.4)	
Clinical diagnosis before baseline coronary angiography, *n* (%)					
Left ventricular ejection fraction <50%, *n* (%)	268 (13.3)	69 (17.6)	115 (12.7)	84 (11.6)	0.01
Extend of CAD at baseline coronary angiography, *n* (%)					
Nonsignificant CAD	248 (12.3)	55 (14.0)	124 (13.7)	69 (9.5)	0.13
Single-vessel disease	569 (28.2)	111 (28.3)	251 (27.8)	207 (28.6)	
Double-vessel disease	549 (27.2)	110 (28.1)	244 (27.0)	195 (26.9)	
Triple-vessel disease	653 (32.3)	116 (29.6)	284 (31.5)	253 (34.9)	
Serum lipids at baseline coronary angiography					
Total cholesterol (mmol L^{-1})	5.1 (1.2)*	5.2 (1.6)	5.1 (1.3)	4.9 (1.1)	<0.001
Low-density lipoprotein cholesterol (mmol L^{-1})	3.1 (1.0)*	3.2 (1.1)	3.1 (1.0)	3.0 (1.0)	<0.001
High-density lipoprotein cholesterol (mmol L^{-1})	1.3 (0.4)*	1.3 (0.4)	1.3 (0.5)	1.3 (0.3)	0.98
Apolipoprotein B (g L^{-1})[‖]	0.85 [0.71 and 1.02]	0.87 [0.73 and 1.06]	0.85 [0.72 and 1.02]	0.82 [0.69 and 1.00]	<0.001
Apolipoprotein A1 (g L^{-1})[‖]	1.27 [1.10 and 1.44]	1.24 [1.09 and 1.44]	1.28 [1.11 and 1.46]	1.26 [1.11 and 1.42]	0.66
Triglycerides (mmol L^{-1})[‖]	1.53 [1.10 and 2.20]	1.59 [1.14 and 2.35]	1.59 [1.10 and 2.20]	1.48 [1.09 and 2.12]	0.01
Inflammation markers and renal function at baseline coronary angiography					
C-reactive protein (mg L^{-1})[‖]	1.65 [0.81 and 3.27]	1.90 [0.96 and 3.69]	1.77 [0.86 and 3.36]	1.34 [0.73 and 2.81]	0.06
eGFR (mL min^{-1})	89.7 (15.4)*	90.6 (17.0)*	89.3 (15.5)*	89.8 (14.3)*	0.57
Medications after baseline coronary angiography, *n* (%)					
Acetylsalicylic acid	1801 (89.2)	347 (88.5)	810 (89.7)	644 (89.0)	0.92
Statins	1783 (88.3)	339 (86.5)	797 (88.3)	647 (89.4)	0.16
β-blockers	1547 (76.6)	310 (79.1)	681 (75.4)	556 (76.8)	0.54
ACE inhibitors and/or ARBs	403 (20.0)	82 (20.0)	174 (19.3)	147 (20.3)	0.48
Loop diuretics	187 (9.3)	49 (12.5)	83 (9.2)	55 (7.6)	0.01

ACE, angiotensin converting enzyme; ARBs, angiotensin receptor blockers; CAD, coronary artery disease; eGFR, estimated glomerular filtration rate.
*Data are the mean (SD) (all such values).
[†]*P*-trend for Healthy Nordic Food Index groups 0–1, 2–3 and 4–6. Calculated with use of linear regression for continuous variables, logistic regression and chi-squared for categorical variables.
[‡]Diabetes, diagnosed or assessed according to baseline serum glucose levels ≥7.0 or a nonfasting glucose ≥11.1 mmol L^{-1}.
[§]Current smoker, based on self-report and cotinine levels >85 (includes ex-smokers <1 month).
[¶]Missing, *n* = 432 (21.4%); 0–1 = 79 (20.4%); 2–3 = 194 (21.5%); 4–6 = 151 (20.9%).
[‖]Median and percentiles [25th and 75th].

expected, high adherence was associated with a higher consumption of dietary fibre ($P < 0.001$), fish ($P < 0.001$), whole grain bread ($P < 0.001$), vegetables ($P < 0.001$) and fruit/ berries ($P < 0.001$). All intake was energy adjusted. A high HNFI score was also associated with a lower E% consumption of total fat ($P < 0.001$), saturated fatty acids (SFA) ($P < 0.001$) and monounsaturated fatty acids ($P < 0.001$), as well as lower intake of processed meat ($P < 0.001$), cheese ($P = 0.002$), butter ($P < 0.001$) and white bread ($P < 0.001$). All food items were energy adjusted (Table 2).

Adherence to the Healthy Nordic diet and outcomes

Median (25th and 75th percentiles) length of follow-up was 10.5 (9.3 and 11.7) years for mortality outcomes and 7.5 (6.3 and 8.7) years for AMI. A total of 171 participants died as a result of cardiovascular disease, 380 died from any cause and 307 patients experienced at least one nonfatal or fatal AMI.

No significant association between adherence to the HNFI and the incidence of total AMI (Table 3) or for nonfatal and fatal AMI separately (see Supporting information, Table S3) was detected. For cardiovascular death, a reduced risk was detected per 1-point increment in the index [HR (95% CI) = 0.90 (0.81–1.01)], although this was statistically insignificant. For all-cause death, a significantly reduced risk association was observed both in the linear estimates [HR (95% CI) = 0.91 (0.84–0.98)] and across adherence categories when comparing the highest versus the lowest adherence group [HR (95% CI) = 0.70 (0.52–0.95)] (Table 3).

Table 2 Baseline daily dietary intake for patients with stable angina pectoris calculated from a food frequency questionnaire

		Healthy Nordic Food Index groups			
Dietary intake	All $n = 2019$	0–1 (lowest) $n = 392$	2–3 $n = 903$	4–6 (highest) $n = 724$	*P*-trend[†]
Energy (MJ [kcal])	8.77 (2.65) [2095 (633)]*	7.69 (2.57) [2095 (633)]	8.48 (2.54) [2095 (633)]	9.71 (2.51) [2095 (633)]	<0.001
Nutrients (E%)[‡]					
Total fat	31.3 (5.4)	33.0 (5.7)	31.3 (5.4)	30.4 (5.0)	<0.001
Saturated and trans fat	11.5 (2.6)	12.5 (2.8)	11.7 (2.6)	10.9 (2.3)	<0.001
Monounsaturated fat	10.1 (1.9)	10.6 (2.0)	10.1 (1.9)	9.8 (1.8)	<0.001
Polyunsaturated fat	7.1 (1.9)	7.2 (2.2)	7.0 (1.9)	7.1 (1.8)	0.7
Protein	16.9 (2.6)	16.3 (2.5)	16.8 (2.6)	17.4 (2.4)	<0.001
Carbohydrate	49.7 (6.4)	48.4 (6.4)	49.8 (6.6)	50.3 (6.0)	<0.001
Dietary fibre[§]	12.2 (3.2)	10.1 (2.7)	12.0 (2.9)	13.6 (3.2)	<0.001
Food items (g day^{-1} 1000 kcal^{-1})					
Fish[¶]	53.5 (28.3)	39.6 (23.3)	51.4 (28.5)	63.7 (26.7)	<0.001
Olive oil	0.28 (0.29)	0.29 (0.33)	0.31 (0.30)	0.33 (0.24)	0.03
Red meat	25.7 (14.1)	26.8 (16.0)	25.3 (13.7)	25.5 (13.5)	0.2
Processed meat	28.8 (15.6)	33.4 (18.1)	29.5 (15.3)	25.4 (13.6)	<0.001
Cheese	13.2 (11.8)	14.4 (13.3)	13.6 (12.2)	12.2 (10.4)	0.002
Milk	152 (109)	162 (125)	151 (109)	148 (98.0)	0.06
Butter and margarine	2.7 (3.9)	3.2 (5.0)	2.8 (3.9)	2.2 (3.1)	<0.001
Egg	8.4 (6.1)	9.3 (7.1)	8.7 (6.3)	7.4 (5.0)	0.06
Potatoes	64.4 (33.8)	64.6 (38.0)	66.4 (35.0)	61.6 (29.3)	<0.001
White bread	37.2 (36.8)	61.7 (40.7)	38.3 (36.4)	22.5 (26.4)	<0.001
Whole grain bread	55.0 (46.7)	32.5 (47.5)	53.8 (47.8)	68.5 (39.3)	<0.001
Vegetables	105 (74.1)	66.0 (48.3)	101 (71.5)	133 (77.9)	<0.001
Fruits and berries	83.5 (63.7)	53.7 (50.1)	81.9 (62.8)	102 (64.9)	<0.001
Cakes	13.4 (12.4)	12.6 (11.9)	13.5 (12.6)	13.9 (12.4)	0.1
Sugar and sweets	4.7 (6.1)	5.4 (6.8)	4.7 (6.2)	4.2 (5.4)	0.001
Alcohol[‖]	1.4 [<0.001 and 2.4]	1.34 [<0.001 and 4.4]	1.43 [<0.001 and 4.1]	1.44 [0.08 and 3.8]	0.02

*Mean (SD) (all such values).

[†]Calculated by using linear regression. *P*-trend for Healthy Nordic Food Index (HNFI) groups 0-1, 2-3 and 4-6.

[‡]E%, percentage of total energy.

[§]Estimated in g day^{-1}1000 kcal^{-1}.

[¶]Includes fish and shellfish.

[‖]Median and percentiles [25th and 75th].

Table 3 Adjusted hazard ratios (HRs) [95% confidence interval (CI)] for acute myocardial infarction (AMI), cardiovascular death and all-cause death according to level of adherence to the Healthy Nordic Food Index (HNFI) for stable angina pectoris patients*

	Events, *n*	Model 1† HR (95% CI)	Model 2‡ HR (95% CI)	Model 3§ HR (95% CI)
AMI	307			
HNFI – linear estimate¶		0.95 (0.86–1.05)	0.97 (0.89–1.05)	0.99 (0.91–1.08)
P for trend		0.32	0.47	0.84
HNFI – categories				
0–1 points (reference)	70	1.00	1.00	1.00
2–3 points	135	0.81 (0.61–1.09)	0.85 (0.63–1.14)	0.88 (0.65–1.18)
4–6 points	102	0.76 (0.56–1.03)	0.83 (0.60–1.14)	0.90 (0.65–1.25)
P for trend‖		0.16	0.28	0.59
Cardiovascular death	171			
HNFI – linear estimate¶		0.85 (0.77–0.94)	0.87 (0.78–0.96)	0.90 (0.81–1.01)
P for trend		0.002	0.009	0.06
HNFI – categories				
0–1 points (reference)	38	1.00	1.00	1.00
2–3 points	84	0.83 (0.56–1.21)	0.86 (0.58–1.26)	0.92 (0.62–1.36)
4–6 points	49	0.56 (0.37–0.86)	0.61 (0.39–0.96)	0.71 (0.45–1.11)
P for trend‖		0.005	0.024	0.17
All-cause death	380			
HNFI – linear estimate¶		0.87 (0.81–0.93)	0.87 (0.81–0.94)	0.91 (0.84–0.98)
P for trend		<0.001	< 0.001	0.009
HNFI – categories				
0–1 points (reference)	84	1.00	1.00	1.00
2–3 points	184	0.83 (0.64–1.08)	0.84 (0.65–1.10)	0.91 (0.70–1.19)
4–6 points	112	0.59 (0.44–0.78)	0.61 (0.45–0.82)	0.70 (0.52–0.95)
P for trend‖		<0.001	0.001	0.017

*CIs were calculated with use of Cox proportional hazards; $n = 2019$.
†Adjusted for age and sex.
‡Adjusted for age, sex and total energy intake.
§Adjusted for age, sex, total energy intake, alcohol intake, diabetes, hypertension, physical activity, current smoking and statin treatment.
¶From linear estimates (per 1-point higher score).
‖From linear analysis, calculated across adherence categories.

In analyses stratified by sex (see Supporting information, Table S4), we observed no significant interaction by sex for AMI. However, there was a significant interaction by sex for cardiovascular ($P = 0.004$) and all-cause death ($P < 0.001$), with stronger protective effects of a high HNFI adherence among women than for men. The crude event-free survival time for each outcome stratified by sex is shown in the Supporting information (Fig. S1). The Kaplan–Meier curves were significantly different only for all-cause death.

Discussion

To our knowledge, this is the first study evaluating the HNFI with respect to clinical outcomes in patients with established CAD. In this prospective cohort study among Norwegian men and women with stable angina pectoris, a higher adherence to the HNFI was statistically significantly associated with a reduced mortality risk. A similar association was detected for cardiovascular death. However, no significant association with the subsequent risk of AMI was detected. For both CVD and total death, we observed a significant modification by sex, with a stronger and more protective effect of a high HNFI adherence among women.

We observed distinct associations between adherence to the HNFI score groups and other dietary habits. In particular, participants with a high adherence to the HNFI had a significantly lower percentage intake of fat and a higher fibre intake than those with a low adherence. Concurrently, they had lower SFA intake as a result of a lower intake of SFA-containing foods such as butter, cheese, milk and processed meat. Although their intake of sugar and sweets was low, they had higher intakes of vegetables, fruit and berries, and whole grain bread, contributing to the high fibre intake in those with a high adherence. These differences in the mentioned food items were expected as a result of the construction of the dietary index [22].

A high intake of whole grains has been reported to be protective against CVD [40]; a high intake of fruits and

berries as well as vegetables has been associated with low CVD and mortality risk [41,42]. Moreover, patients with a high HNFI also had lower blood lipids despite similar exposure to statins. A low intake of SFA was one of the most obvious dietary differences between patients with high versus low adherence. In addition, participants with a high adherence had a higher intake of fish, as also associated with low CVD risk and low mortality risk [3,43].

Participants with a high versus a low adherence to the HNFI were different with regard to several cardiovascular risk factors and lifestyle characteristics. They were less likely to be smokers and were physically more active than those with a low adherence. The latter may explain why participants with a high adherence had a body mass index similar to those with a low adherence despite a higher energy intake. A similar finding was reported by Roswall *et al.* [22] in healthy participants. Because data on physical activity were self-reported, it was not possible to calculate the energy cost of these extra activities. However, this highlights the importance of physical activity being associated with low mortality [44–46].

To summarise, positive associations with lifestyle factors in combination with a healthy diet may explain the observed inverse association between HNFI adherence and cardiovascular and total mortality. However, the lack of any effect of the HNFI on AMI occurrence is difficult to explain, although it may be a result of the extensive treatment with statins. There was limited power to detect significant differential associations between HNFI and the outcomes fatal and nonfatal AMI separately because of a low number of fatal AMIs.

Our results for outcomes of cardiovascular death and all-cause death are in line with several other studies on the HNFI conducted mainly in healthy subjects [14,32,47]. However, our findings with respect to the outcomes of AMI are in contrast to those of a recent Danish study based on a healthy population, in which it was shown that high adherence to the HNFI was associated with a lower risk of myocardial infarction among both men and women [23].

Compared to the HNFI, there is ample evidence that a Mediterranean Diet score is protective against CVD morbidity and mortality primarily in the general population. Studies of the Mediterranean Diet have been conducted in several settings around the world [48–51] and have mostly reported significant reductions in risk of coronary heart disease [10], CVD progression [52], AMI [50] and mortality [13]. A recent review by Mattioli *et al.* [52] declared that there is insufficient evidence for any clear positive effects as a result of following a Mediterranean Diet on CVD outcomes among patients with established coronary heart disease.

By contrast to the HNFI, the Mediterranean Diet score includes nine food groups and nutrients that are either positively or negatively associated with health effects [53]. Some of these food groups, which are traditionally highly consumed in the Mediterranean areas, in particular, olive oil and legumes, are consumed at very low levels in the Nordic countries (compare Table 2 for olive oil intake in our population). In comparison to the Mediterranean index, the HNFI may capture only partly a Healthy Nordic diet because it does not contain any information on intake of meat and edible oils. Thus, the absence of certain food items or nutrients that contribute to energy intake clearly features some limitations with respect to this index. This might explain why we did not identify any association between HNFI adherence and AMI.

Nutraceuticals have been described as useful compounds that are able to reduce the overall cardiovascular risk caused by dyslipidaemia by acting in parallel to statins or as adjuvants in the case of failure or in situations where statins cannot be used [54]. However, the underlying mechanisms are not fully understood but may be related to reducing 7a-hydroxylase, decreasing 3-hydroxy-3-methylglutaryl-CoA reductase mRNA levels, reducing the secretion of very low-density lipoprotein or increasing the faecal excretion of cholesterol [54]. As described by Scicchitano *et al.* [54], nutraceuticals are presented at high levels in fish, fruits and vegetables. Because these food items form the basis for the HNFI (fish, apples and pears, root vegetables and cabbages), they may possibly play a part in the associations between the HNFI and outcomes. Future studies should thus explore whether risk associations of the HNFI may be mediated by nutraceuticals.

One particularly strong feature of the present study was the inclusion of both men and women. We observed a significantly stronger inverse association between the HNFI and mortality in women compared to men. This should motivate future studies on sex differences of dietary patterns in relation to CVD development. Moreover, linkage to the CVDNOR project and the Cause of Death registry assured an almost complete follow-up. Other strengths included detailed information on potential confounders such as lifestyle, cardiovascular history and the results from cardiac examinations, as well as medication. However, the information on lifestyle was self-reported and information bias cannot be ruled out. Use of the FFQ is easy, even for large populations, in that it requires simple administration, has a rather low respondent burden and may capture foods eaten rarely.

Some limitations of the present study also need to be addressed. Dietary intake was only assessed at baseline, and dietary habits may have changed during the long follow-up period and not been captured by the analysis. In

general, data obtained from the FFQ have the potential to be limited by the foods included, as well as by misclassification of dietary intake and estimation of portion sizes. The FFQ also has less sensitivity compared to a 24-h dietary recall [55]. However, to capture the usual diet, more than one 24-h diet recall would have been required, which was by far beyond the capacity of the study centre. Because our study originally included more than 3000 patients, administering a FFQ is the only method for obtaining valid data on habitual diet. This is in line with other studies on dietary patterns, including the Mediterranean diet amongst others [13,14,22,23]. In addition, the FFQ that was used has been validated previously [28–31]. Furthermore, the willingness of the participants to provide information on a realistic dietary consumption can be biased; for example, obese individuals and especially women often under-report their total energy intake [56]. Because the majority of participants were men (81%), this factor may be less important for the present study. Notably, the strongest risk associations were observed among women, suggesting that misclassification of data relevant to the HNFI was not a greater problem in women. Furthermore, we observed no association between the HNFI and waist circumference, which therefore did not materially influence our results. Because our study started before the HNFI was developed [14], the FFQ used in the present study was not specifically developed for use of the HNFI index. This is illustrated by the replacement of rye bread in the original index by whole grain bread in our analysis. Certainly, rye bread is common in Denmark but not in Norway [57–59].

We only had self-reported data on physical activity and a larger proportion (21.4%) had missing information. Furthermore, self-reported data on physical activity may not be accurate and reliable and have been shown to result in both higher and lower levels of physical activity than actually measured directly [60]. Thus, residual confounding because of inadequate data ragarding physical activity cannot be excluded. Finally, the potential effect modification by physical activity should be evaluated in future studies. Data on socio-economic status were not available for the majority of the study participants and this might have increased the probability for residual confounding by non-nutritional factors.

Because the present study cohort consisted mainly of elderly male patients who had established atherosclerotic disease and were therefore being treated with various medications, the results may not be generalised to a younger or a healthy, general population.

In conclusion, adherence to the HNFI was associated with low risk of mortality among patients with stable angina pectoris. There was no significant association with risk of AMI. This highlights the importance of dietary habits in this patient group, with other dietary patterns besides the Mediterranean Diet possibly having positive health effects in these patients. However, the HNFI may need further refinement to provide a more representative dietary profile.

Transparency statement

The lead author affirms that this article is an honest, accurate and transparent account of the study being reported, that no important aspects of the study have been omitted and that any discrepancies from the study as planned have been explained. The reporting of this work is compliant with STROBE guidelines. The trial has been registered at https://www.clinicaltrials.gov/ as NCT 00354081.

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Conflicts of interest, source of funding and authorship

The authors declare that they have no conflicts of interest.
This study was funded by the Norwegian Health Association of Oslo in Norway.
NGP, JD and ON designed the study. JD, ES, TK, VL and ON supervised the study. NGP and JA conducted the statistical analysis. KV and CAD provided the nutritional data. GST furnished the study endpoints. All authors critically revised the manuscript and approved the final version submitted for publication.

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Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Figure S1. Kaplan–Meier survival curves for endpoints acute myocardial infarction (AMI), cardiovascular death and all-cause death for Healthy Nordic Food Index (HNFI) categories, stratified by sex.

Table S1. Daily dietary (g day^{-1}) intake of food groups included in the Healthy Nordic Food Index.

Table S2. Healthy Nordic Food Index score distribution as a percentage for a 1-point higher score, presented for the total population, men and women.

Table S3. Adjusted hazard ratios (95% confidence interval) for nonfatal and fatal acute myocardial infarction, according to level of adherence to the Healthy Nordic Food Index in patients with stable angina pectoris.

Table S4. Adjusted hazard ratios (95% confidence interval) for acute myocardial infarction, cardiovascular death and all-cause death according to level of adherence to the Healthy Nordic Food Index in patients with stable angina pectoris.