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FULL RESEARCH PAPER
Cardiovascular Disease

The DASH diet is associated with a lower risk of heart failure: a cohort study

Daniel B. Ibsen [1,2]*, Emily B. Levitan[3], Agneta Åkesson [1], Bruna Gigante[4], and Alicja Wolk[1,5]

[1]Unit of Cardiovascular and Nutritional Epidemiology, Institute of Environmental Medicine, Karolinska Institutet, Nobels Väg 13, Stockholm 171 77, Sweden; [2]Research Unit for Epidemiology, Department of Public Health, Aarhus University, Bartholins Allé 2, 8000 Aarhus C, Denmark; [3]Department of Epidemiology, University of Alabama at Birmingham, Ryals Public Health Building (RPHB), 1665 University Boulevard, Birmingham, Alabama 35233, USA; [4]Division of Cardiovascular Medicine, Department of Medicine, Karolinska Institutet, Solna Karolinska University Hospital D1:04, 171 76 Stockholm, Sweden; and [5]Unit of Medical Epidemiology, Department of Surgical Sciences, Uppsala University, Akademiska sjukhuset ingång 78, 751 85 Uppsala, Sweden

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Aims

Trials demonstrate that following the DASH diet lowers blood pressure, which may prevent the development of heart failure (HF). We investigated the association between long-term adherence to the DASH diet and food substitutions within the DASH diet on the risk of HF.

Methods and results

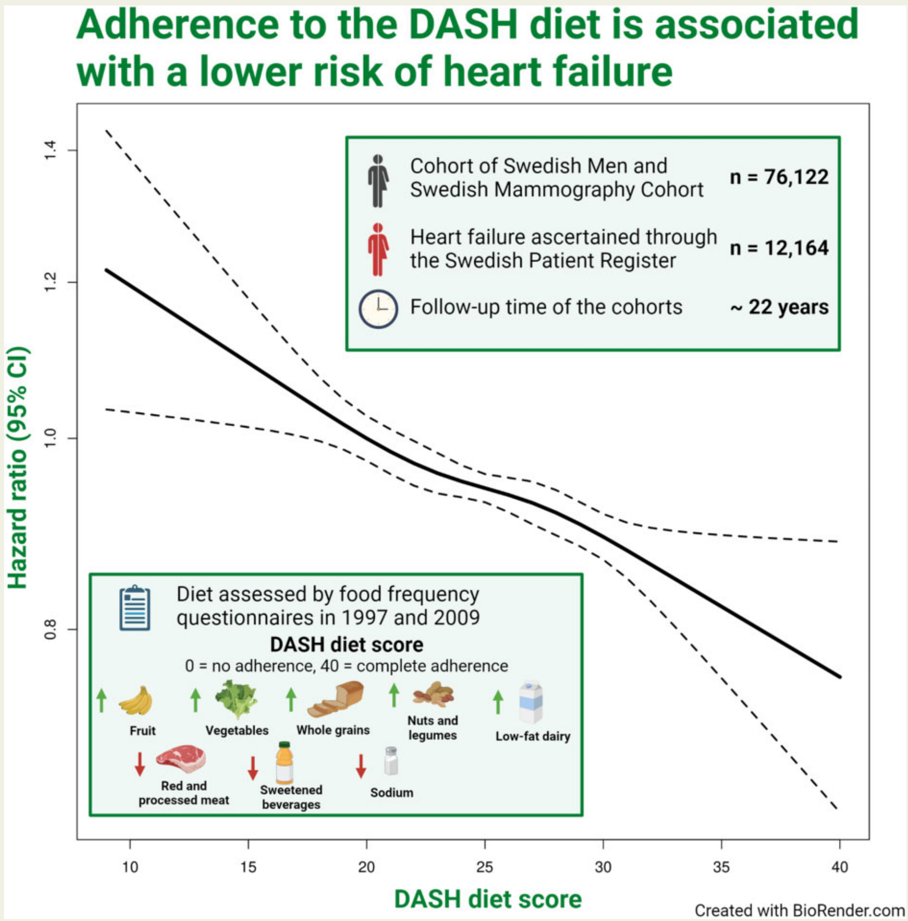
Men and women aged 45–83 years without previous HF, ischaemic heart disease or cancer at baseline in 1998 from the Cohort of Swedish Men ($n = 41\ 118$) and the Swedish Mammography Cohort ($n = 35\ 004$) were studied. The DASH diet emphasizes intake of fruit, vegetables, whole grains, nuts and legumes, and low-fat dairy and deemphasizes red and processed meat, sugar-sweetened beverages, and sodium. DASH diet scores were calculated based on diet assessed by food frequency questionnaires in late 1997 and 2009. Incidence of HF was ascertained using the Swedish Patient Register. Multivariable Cox proportional hazards models were used to estimate hazard ratios (HRs) with 95% confidence intervals (CIs). During the median 22 years of follow-up (1998–2019), 12 164 participants developed HF. Those with the greatest adherence to the DASH diet had a lower risk of HF compared to those with the lowest adherence (HR 0.85, 95% CI: 0.80, 0.91 for baseline diet and HR 0.83, 95% CI: 0.78, 0.89 for long-term diet, comparing quintiles). Replacing 1 serving/day of red and processed meat with emphasized DASH diet foods was associated with an 8–12% lower risk of HF.

Conclusion

Long-term adherence to the DASH diet and relevant food substitutions within the DASH diet were associated with a lower risk of HF.

* Corresponding author. Tel: +45 28835317, Email: daniel.ibsen@ki.se

Graphical Abstract



Introduction

Heart failure (HF) is an immense public health problem, with an estimated global incidence of 10–90 cases/10 000 person-years, and is the number one cause of hospitalization among the elderly in high-income countries.[1] Hence, prevention of HF has a high priority.

Adherence to healthy dietary patterns, as defined by national dietary guidelines, including the Nordic Nutrition Recommendations used in Sweden, and the Mediterranean diet, has been associated with the lower risk of all-cause mortality, cardiovascular mortality, and incidence of HF.[2–5] In randomized controlled trials (RCTs), consuming a healthy diet has also been shown to improve key risk factors for the development of HF including high blood pressure, high body mass index (BMI), high blood glucose, and LDL-cholesterol.[6] In particular, strong evidence from RCTs has shown that the Dietary Approach to Stop Hypertension (DASH) diet, recommending higher intakes of fruits, vegetables, whole grains, nuts and legumes, and low-fat dairy and lower intakes of red and processed meat, sugar-sweetened beverages, and sodium, lowered blood pressure.[6] While sodium reduction in patients with HF is controversial, there is good evidence for its role in the prevention of HF, particularly in high-risk populations.[7]

Concerning the role of the DASH diet in the prevention of HF, evidence from US cohorts has been conflicting. A higher HF risk was observed among those with the greatest adherence to the DASH diet in the Cardiovascular Health Study[8] whereas a lower risk was reported in the Multi-Ethnic Study of Atherosclerosis[9] and the Reasons for Geographic And Racial Differences in Stroke cohort.[10] We have previously observed that adherence to the DASH diet was associated with lower HF risk in women[11] and men,[12] but these studies examined baseline diet only and had a shorter follow-up time. Updating these analyses would improve our understanding of the long-term impact of adhering to the DASH diet on HF risk. In addition, several cohort studies have investigated individual components included in the DASH diet (e.g. vegetables) in relation to HF; however, none has specified food substitutions, which provide a clearer interpretation of the association of specific food items with health outcomes and direct simple actionable dietary shifts.[13] One important food group is red and processed meat as it contains detrimental (e.g. sodium in processed meat) but also beneficial nutrients (e.g. U-shaped relation for iron status[14]) for the development of HF.

Our main hypothesis was that long-term adherence to the DASH diet is associated with a lower risk of HF in middle-aged and elderly adults. We also examined the association between relevant food substitutions among DASH diet foods and risk of HF.

Methods

Ethical approval

The study complies with the Declaration of Helsinki and was approved by the Regional Ethical Review Board at Karolinska Institute, Stockholm, Sweden. Completion and return of the questionnaires was considered consent. The two population-based cohorts from central Sweden, the Cohort of Swedish Men (COSM) and the Swedish Mammography Cohort (SMC), were registered at ClinicalTrials.gov with identifiers NCT01127711 and NCT01127698, respectively, and both cohorts belong to the national research infrastructure SIMPLER (www.simpler4health.se).

Study population

We used self-reported questionnaire-based information on demographics, dietary intake, and lifestyle obtained in late 1997 and in 2009 in both COSM and SMC. The COSM includes 48 850 men born between 1918 and 1952 (49% of invited). The SMC includes 39 984 women born 1914–48 (70% of invited). In 2009, an updated questionnaire was sent to participants in the COSM ($n = 29\ 068$ responded, 90% of invited) and SMC ($n = 30\ 134$ responded, 84% of invited) who were still alive and residing in the study areas.[15] We started follow-up of these cohorts on 1 January 1998 and, at baseline, responders were comparable to the general Swedish population regarding the distribution of age, education, and BMI.[15]

We excluded participants with missing or incorrect ID or a diagnosis of cancer ($n = 5770$) and further excluded participants with HF ($n = 1268$) or ischaemic heart disease ($n = 4710$) before the baseline. Those with cancer were excluded due to change in health status and likely in health behaviour. Participants with previous ischaemic heart disease were also excluded because of the conditions' strong relationship with the development of HF and our focus on prevention in the general population. To improve the quality of reported intakes, we additionally excluded implausible total energy intakes (±3 SD of mean total energy intake on the log scale) ($n = 964$) (Supplementary material online, *Figures S1 and S2*). A total of 76 122 participants were included in the analysis using the baseline data.

Diet assessments

Dietary intake in late 1997 was assessed using a 96-item food frequency questionnaire (FFQ) that asked participants about their average intake of foods and meals during the last 12 months.[15,16] Response categories ranged from 'never' to '≥3 times/day' for 88 items and 8 items were open-ended questions. Age-specific portion sizes together with information from the Swedish Food Administration nutrient composition database[17] were used to estimate average intake of foods and nutrients for each participant. The follow-up FFQ in 2009 was extended to 132 food items to include new foods on the market and usual foods consumed at the time.[15]

DASH diet

We used the score proposed by Fung *et al.*[18] to calculate adherence to the DASH diet. The diet score includes eight dietary components: fruits, vegetables, nuts and legumes, low-fat dairy, whole grains, sodium, sweetened beverages, and red and processed meat. Supplementary material online, *Table S1* shows which foods were included in each category. For each component, participants are categorized into quintiles according to the amount of intake of the specific item and scored 1–5 points based on the quintile they are in. The total score is the sum of points (maximum 40) across the dietary components. We calculated the score in men and women combined. Correlations between the FFQ-based estimates and four 1-week diet records distributed over 1 year were 0.5–0.7 for fruits,[19] 0.4–0.6 for vegetables,[19] 0.5–0.7 for whole grains,[20] 0.4–0.6 for dairy,[21] 0.3–0.7 for red and processed meats,[22] and 0.6 for sweetened beverages.[23]

Heart failure ascertainment and follow-up of health status

HF incidence was ascertained using the Swedish Patient Register (inpatient and outpatient specialist care) and defined according to the International Classification of Disease-10 (ICD-10) codes I50 and I11.0, listed as either primary diagnosis or at any diagnosis position. Diagnosis of HF in the Register was previously validated against review of medical records using the European Cardiology Society criteria for HF and had been found that 95% of patients with HF as primary diagnosis code and 76% with HF as secondary diagnosis code were correctly classified.[24] Dates of death were obtained from the Swedish Death Register.

Assessment of covariates

Information on education, weight, height, minutes per day walking/cycling, use of aspirin, corticosteroids, dietary supplements, use of hormone replacement therapy (women), alcohol, smoking status, history of hypercholesterolaemia, hypertension, and diabetes, and family history of myocardial infarction was self-reported in the baseline questionnaire. Atrial fibrillation ICD-10 code I48 was ascertained by linkage to the Swedish Patient Register.

Statistical analyses

To estimate the association, hazard ratio (HR) and 95% confidence intervals (CIs), between adherence to the DASH diet and risk of HF, we used Cox proportional hazards regression with age as the underlying time scale. Participants were followed up from January 1, 1998, to the date of diagnosis of HF, death, or end of follow-up December 31, 2019, whichever came first. Adherence to the DASH diet was modelled as quintiles of the DASH diet score in men and women combined because results were similar in analyses when DASH diet scores were sex specific (P for interaction = 0.56). Potential confounders were selected based on a review of the literature on risk factors for the development of HF and the use of directed acyclic graphs to show assumed relationship between variables (Supplementary material online, *Figure S3*). The basic model was adjusted for age and sex (men, women). The multivariable model was adjusted for potential confounders including education (primary, high school, university), smoking (never; ex-smoker <20, 20–39, or ≥40 pack-years; current <20, 20–39, or ≥40 pack-years), alcohol (g/day, *continuous*), walking/cycling (<20, 20–40, 40–60, or >60 min/day), corticosteroid use (yes, no), aspirin use (yes, no), dietary supplement use (regularly, sometimes, no), use of hormone replacement therapy in women (yes, no), family history of myocardial infarction (yes, no), and total energy intake (kcal/day, *continuous*). The model was further adjusted for BMI (≤18.5, 18.5–24.9, 25–29.9, >30 kg/m^2), hypertension (yes, no), hypercholesterolaemia (yes, no), diabetes (yes, no), and atrial fibrillation (yes, no). Due to few missing values in the included variables, they were indicated with their own 'missing' category in the main analysis. To investigate the association with long-term adherence, we updated the dietary intake to the average intake of the two FFQs among those who had completed the follow-up FFQ in 2009. Baseline intake was used for participants who were censored before 2009 or did not complete the FFQ in 2009. The assumption of proportional hazards was assessed using log–log plots and no evidence for violation of the assumption was detected. The association between adherence to the DASH diet and risk of HF was also modelled using a restricted cubic spline function with four knots of the DASH diet score.

We estimated the association related to food substitutions between foods within the DASH diet. One serving/day of red and processed meat (100 g/day) was replaced with 1 serving/day of fruit (100 g/day), vegetables (100 g/day), nuts and legumes (20 g/day), whole grains (100 g/day), or low-fat dairy (200 g/day). The food substitution was obtained by including all DASH diet foods (1 serving/day, continuous) separately in the statistical model, except for the food to be replaced (here red and processed meat), and a variable that is the sum of all the foods included in the DASH diet score.[13] As the total intake of all DASH diet score foods is fixed in the model, a higher intake of each of the included foods must be at the expense of the food not included in the model (here red and processed meat). In other words, participants with a higher intake of fruit and a lower intake of red and processed meat are compared with participants with a lower intake of fruit and a higher intake of red and processed meat. Because the energy density of the substituted foods is not equal, there is a residual energy substitution from foods not included in the model. As a sensitivity analysis, we estimated the food substitutions without adjustment for total energy intake. We also investigated the association between each of the food groups (i.e. sodium not included) in the DASH diet separately, adjusting for the other food groups on risk of HF. Sodium, as the only nutrient, was investigated separately. Each item was modelled using a restricted cubic spline function with four knots.

Potential effect modification by self-reported history of hypertension and history of diabetes was also assessed in stratified analyses. To estimate the public health benefit on the HF risk of adhering to the DASH diet, assuming a causal relationship, we used the additive Cox model[25] by estimating multivariable adjusted rate differences and corresponding 95% CIs at 20 years of follow-up.

In sensitivity analyses, we used multiple imputation with chained equations to impute five datasets in which we re-ran the main analysis and subsequently pooled the results because of missing data (<0–11% missing) on some of the included variables. We also further excluded those with a high risk of HF at baseline, i.e. those with hypertension, diabetes, and/or atrial fibrillation, to avoid bias due to underlying illnesses that could have influenced dietary intake. We re-ran the main analysis including only those with HF as the main diagnosis code to investigate potential outcome misclassification. As anti-cancer treatment may induce HF,[26] we censored all participants who developed any type of cancer before development of HF. Incidence of cancer was obtained from the Swedish Cancer Register. Lastly, because of the built-in selection bias of the HR,[27] we truncated the end of follow-up date at 5, 10, 15, and 20 years of follow-up to investigate the stability of HRs over time.

All analyses were conducted using R (R Core Team, Vienna, Austria) version 3.6.0. The data underlying this article cannot be shared publicly because the data are classified as sensitive data under the European General Data Protection Regulation. The data and underlying code can be shared on reasonable request to the corresponding author through the Swedish National Research Infrastructure SIMPLER (www.simpler4health.se).

Results

Baseline characteristics

Participants with the greatest adherence to the DASH diet (score 30–40 points) generally had healthier characteristics at the baseline than those with the lowest adherence (score 8–21 points) (*Table 1*). Those with the greatest adherence appear to be older, be women, and have a normal BMI, a longer education, never smoked, a lower intake of alcohol, and a less physical inactivity and to take dietary supplements. The median follow-up time was 22 years and during 6 001 289 person-years of observation 12 164 participants developed HF [as primary diagnosis $n = 4609$ (2625 men, 1984 women), or at any other diagnosis position $n = 7555$ (3963 men, 3592 women)]. Of all men, 7% were in the highest and 35% in the lowest category of adherence to the DASH diet, corresponding numbers for women were 26% and 10%.

Adherence to the DASH diet and risk of HF

After multivariable adjustment (*Table 2*, multivariable adjusted 1), those with the greatest adherence to the DASH diet at baseline (HR 0.85, 95% CI: 0.80, 0.91) and long-term adherence (HR 0.83, 95% CI: 0.78, 0.89) had a lower risk of HF compared to those with the lowest adherence. Results were similar after further adjustment for BMI, hypertension, hypercholesterolaemia, diabetes, and atrial fibrillation at baseline. When adherence to the DASH diet was modelled as restricted cubic splines, a clear linear inverse association was observed with greater adherence to the DASH diet (*Figure 1*). For each 2 standard deviations higher DASH diet score (9.1 points), a lower risk of HF was observed (HR 0.89, 95% CI: 0.86, 0.93). We estimated that, under the assumption of a causal effect, about 19 cases (95% CI: 13, 25) of HF could have been prevented for every 10 000 person-years if all had the greatest adherence to the DASH diet (*Table 3*). In stratified analyses, there was not any clear effect modification by previous diabetes or hypertension (Supplementary material online, *Figures S4* and *S5*).

Food substitutions and risk of HF

Replacing 1 serving/day of red and processed meat with 1 serving/day of fruits, vegetables, nuts and legumes, low-fat dairy, or whole grains was associated with ∼8–12% lower risk of HF after multivariable adjustment (*Figure 2*). Similar associations were observed after adjustment for BMI, hypertension, hypercholesterolaemia, diabetes, and atrial fibrillation at baseline (Supplementary material online, *Figure S6*) and when the model was not adjusted for total energy intake (Supplementary material online, *Figures S7* and *S8*).

Individual DASH diet components and risk of HF

Non-linear associations were observed for the intake of low-fat dairy and red and processed meat (*Figure 3*). For low-fat dairy, the lowest risk of HF, compared with no intake, was observed at around 150 g/day ($p_{\text{non-linear trend}} = 0.002$). For red and processed meat, the lowest risk of HF, compared with no intake, was observed around 50 g/day ($P_{\text{non-linear trend}} < 0.001$). In contrast, a linear relation was observed for sweetened beverages ($P_{\text{non-linear trend}} = 0.40$) and for sodium ($P_{\text{non-linear trend}} = 0.51$) (*Figure 3*). Linear relationships were also observed for remaining component (Supplementary material online, *Figure S9*).

Table 1 Baseline characteristics of participants from the Cohort of Swedish Men and Swedish Mammography Cohort 1997 examination according to quintiles of Dietary Approach to Stop Hypertension diet score adherence

Characteristics	8–21 points (*n* = 18 027), median (10th, 90th), *n* (%)	22–24 points (*n* = 17 620), median (10th, 90th), *n* (%)	25–26 points (*n* = 12 541), median (10th, 90th), *n* (%)	27–29 points (*n* = 15 721), median (10th, 90th), *n* (%)	30–40 points (*n* = 12 213), median (10th, 90th), *n* (%)	Overall (*n* = 76 122), median (10th, 90th), *n* (%)
DASH diet score (points)	19 (16, 21)	23 (22, 24)	26 (25, 26)	28 (27, 29)	31 (30, 34)	25 (19, 31)
Age (years)	57 (48, 74)	59 (49, 75)	59 (49, 75)	60 (50, 74)	60 (50, 74)	59 (49, 74)
Sex (women)	3629 (20%)	6667 (38%)	6069 (48%)	9352 (59%)	9287 (76%)	35004 (46%)
BMI (kg/m^2)						
Normal weight (18.6–24.9)	7610 (42%)	8008 (45%)	5987 (48%)	7878 (50%)	6629 (54%)	36112 (47%)
Missing	908 (5%)	671 (4%)	421 (3%)	406 (3%)	225 (2%)	2631 (3%)
Education						
University	2451 (14%)	2718 (15%)	2318 (18%)	3191 (20%)	2972 (24%)	13650 (18%)
Missing	72 (0%)	72 (0%)	46 (0%)	62 (0%)	48 (0%)	300 (0%)
Smoking (pack-years)						
Never	6259 (35%)	7208 (41%)	5672 (45%)	7766 (49%)	6605 (54%)	33510 (44%)
Missing	1133 (6%)	985 (6%)	651 (5%)	725 (5%)	506 (4%)	4000 (5%)
Alcohol (g/day)	6.2 (0.0, 21)	5.4 (0.0, 19)	5.1 (0.0, 18)	4.5 (0.0, 17)	3.5 (0.0, 14)	5.0 (0.0, 18)
Walking and cycling (min/day)						
Never/seldom	2870 (16%)	2120 (12%)	1212 (10%)	1325 (8%)	789 (6%)	8316 (11%)
Missing	2014 (11%)	1677 (10%)	1020 (8%)	1133 (7%)	768 (6%)	6612 (9%)
Aspirin use (yes)	10281 (57%)	9487 (54%)	6516 (52%)	7977 (51%)	5993 (49%)	40254 (53%)
Missing	1873 (10%)	1970 (11%)	1475 (12%)	1746 (11%)	1424 (12%)	8488 (11%)
Corticosteroid use (yes)	1121 (6%)	1115 (6%)	857 (7%)	1171 (7%)	889 (7%)	5153 (7%)
Missing	1580 (9%)	1820 (10%)	1330 (11%)	1808 (12%)	1553 (13%)	8091 (11%)
Dietary supplement use (regularly)	2062 (11%)	2675 (15%)	2292 (18%)	3367 (21%)	3318 (27%)	13714 (18%)
Missing	1320 (7%)	1323 (8%)	870 (7%)	1025 (7%)	748 (6%)	5286 (7%)
HRT use[a] (yes)	1422 (39%)	2831 (42%)	2724 (45%)	4450 (48%)	4613 (50%)	16040 (46%)
Missing	167 (5%)	260 (4%)	185 (3%)	294 (3%)	266 (3%)	1172 (3%)
History of diabetes (yes)	735 (4%)	833 (5%)	619 (5%)	802 (5%)	598 (5%)	3587 (5%)
History of hypertension (yes)	3639 (20%)	3636 (21%)	2625 (21%)	3252 (21%)	2473 (20%)	15625 (21%)
History of hypercholesterolaemia (yes)	1786 (10%)	1732 (10%)	1165 (9%)	1545 (10%)	1167 (10%)	7395 (10%)
History of atrial fibrillation (yes)	271 (2%)	254 (1%)	192 (2%)	212 (1%)	162 (1%)	1091 (1%)
Family history of MI (yes)	497 (3%)	521 (3%)	356 (3%)	520 (3%)	435 (4%)	2329 (3%)
Total energy intake (kJ/day)	2200 (1300, 3400)	2100 (1200, 3400)	2100 (1300, 3400)	2000 (1300, 3400)	2000 (1400, 3200)	2100 (1300, 3400)
Fruits (g/day)	67 (19, 150)	100 (33, 220)	140 (55, 290)	180 (85, 350)	260 (130, 460)	130 (37, 320)
Vegetables (g/day)	82 (28, 160)	120 (47, 220)	150 (68, 270)	180 (92, 320)	230 (130, 400)	140 (51, 290)
Nuts and legumes (g/day)	24 (0.0, 65)	26 (3.2, 77)	29 (4.8, 83)	35 (11, 90)	49 (19, 120)	28 (3.5, 83)
Low-fat dairy products (g/day)	19 (0.0, 440)	150 (0.0, 600)	210 (0.0, 660)	260 (0.0, 690)	320 (61, 750)	190 (0.0, 650)
Whole grains (g/day)	90 (17, 250)	120 (38, 310)	140 (45, 330)	140 (53, 330)	160 (67, 330)	130 (39, 320)
Sodium (mg/day)	3600 (2500, 4300)	3400 (2300, 4200)	3100 (2200, 4200)	2800 (2200, 4100)	2600 (2200, 3800)	3200 (2200, 4200)
Sweetened beverages (g/day)	140 (0.0, 660)	31 (0.0, 510)	0.0 (0.0, 330)	0.0 (0.0, 260)	0.0 (0.0, 140)	0.0 (0.0, 430)
Red and processed meat (g/day)	100 (47, 160)	84 (35, 150)	77 (33, 140)	69 (30, 130)	56 (20, 99)	78 (31, 140)

BMI, body mass index; DASH, Dietary Approach to Stop Hypertension; HRT, hormone replacement therapy; MI, myocardial infarction.
[a]In women only (*n* = 35 004).

Sensitivity analyses

The pattern of associations was similar to the main analysis after including only those with a primary diagnosis code for HF (*Table 2*), using multiple imputation for missing variables (HR 0.86, 95% CI: 0.80, 0.91 for baseline comparing the highest to lowest adherence group) or excluding those at high risk of HF at baseline (HR 0.80, 95% CI: 0.73, 0.87 for baseline comparing the highest to lowest adherence group). In total, 2121 participants

Table 2 Hazard ratios and 95% confidence intervals for the association between adherence to the Dietary Approach to Stop Hypertension diet score and risk of heart failure (n = 76 122, n HF cases = 12 164, person-years = 6 001 289)

	DASH diet score				
	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5
Range	8–21	22–24	25–26	27–29	30–40
Cases	2881	2908	2089	2482	1804
Person-years	1 391 820	1 384 579	992 371	1 252 345	980 174
HR (95% CI)					
Baseline diet					
Age and sex adjusted[a]	1.00 (ref)	0.93 (0.88, 0.98)	0.90 (0.85, 0.95)	0.82 (0.78, 0.87)	0.76 (0.71, 0.81)
Multivariable adjusted 1[b]	1.00 (ref)	0.96 (0.91, 1.01)	0.96 (0.91, 1.02)	0.90 (0.85, 0.95)	0.85 (0.80, 0.91)
Multivariable adjusted 2[c]	1.00 (ref)	0.94 (0.90, 0.99)	0.94 (0.89, 1.00)	0.87 (0.82, 0.92)	0.83 (0.78, 0.88)
Long-term diet					
Age and sex adjusted	1.00 (ref)	0.87 (0.83, 0.92)	0.81 (0.77, 0.86)	0.79 (0.74, 0.83)	0.74 (0.69, 0.78)
Multivariable adjusted 1	1.00 (ref)	0.91 (0.87, 0.96)	0.87 (0.82, 0.92)	0.86 (0.81, 0.91)	0.83 (0.78, 0.89)
Multivariable adjusted 2	1.00 (ref)	0.90 (0.86, 0.95)	0.86 (0.81, 0.91)	0.84 (0.80, 0.89)	0.82 (0.77, 0.87)
Primary HF diagnosis only[d]					
Cases	1124	1116	756	925	688
Person-years	1 254 117	1 241 648	885 081	1 126 044	889 236
Age and sex adjusted	1.00 (ref)	0.93 (0.86, 1.01)	0.87 (0.79, 0.95)	0.82 (0.75, 0.90)	0.77 (0.70, 0.86)
Multivariable adjusted 1	1.00 (ref)	0.97 (0.89, 1.05)	0.93 (0.85, 1.02)	0.90 (0.82, 0.98)	0.88 (0.79, 0.98)
Multivariable adjusted 2	1.00 (ref)	0.95 (0.87, 1.03)	0.91 (0.83, 1.00)	0.86 (0.79, 0.95)	0.85 (0.77, 0.95)

BMI, body mass index; CI, confidence interval; DASH, Dietary Approach to Stop Hypertension; HF, heart failure; HR, hazard ratio.
[a]Age and sex adjusted: age, sex.
[b]Multivariable adjusted 1: age, sex, education, smoking, alcohol, walking/cycling, corticosteroid use, aspirin use, dietary supplements use, use of hormone replacement therapy in women, family history of myocardial infarction, total energy intake.
[c]Multivariable adjusted 2: multivariable adjusted 1, BMI, history of hypertension, hypercholesterolaemia, diabetes and atrial fibrillation at baseline.
[d]n = 68 567, n HF cases = 4609, baseline diet.

developed cancer before HF. Associations were similar after censoring participants who developed cancer before HF [HR (95% CI), baseline diet; for multivariable model 2: 0.84 (0.78, 0.90); multivariable model 3: 0.81 (0.77, 0.87); comparing the highest to lowest adherence group]. During the follow-up period, HRs were attenuated over time when comparing those with the highest to the lowest adherence to the DASH diet using multivariate model 1 and stopping the follow-up time after 5, 10, 15, and 20 years of follow-up [corresponding HRs (95% CIs) were: 0.77 (0.63, 0.93), 0.77 (0.68, 0.87), 0.83 (0.78, 0.90), 0.85 (0.80, 0.92)].

Discussion

In this cohort of Swedish middle-aged and elderly adults, we found that greater adherence to the DASH diet was associated with a lower risk of HF. Replacing 1 serving/day of red meat with 1 serving/day of fruits, vegetables, nuts, and legumes, low-fat dairy, or whole grains was also associated with an 8–12% lower risk of HF, although investigation into individual foods suggested that low intakes of low-fat dairy (around 150 g/day) and red meat (around 50 g/day) may be associated with a lower risk whereas higher intakes were associated with a higher risk of HF, compared with no intake.

Strengths and limitations

Strengths of this study include the prospective and population-based design, nearly complete follow-up via linkage to national health registries with high validity of the HF diagnoses and assessment of long-term dietary intakes. Dietary intake was measured using self-reported FFQs and, although the FFQ showed reasonable correlations compared with four 1-week diet records distributed over a 1-year period, measurement error is present. Due to the design, this measurement error is, however, unlikely to be related to the outcome and, therefore, most likely to have attenuated our results. Indeed, when we updated the dietary information among those who answered the second FFQ in 2009, using a better measure of long-term dietary intake, the associations were slightly stronger indicating that bias towards the null was present. The sensitivity analyses did not indicate substantial bias from misclassification of HF diagnosis, reverse causation, missing data or by HF induced by anti-cancer treatments, as results were in the same direction as the main analysis. HRs were attenuated over the follow-up period, indicating an effect of the built-in selection bias of the HR; likely in combination of the misclassification of dietary intake over time. The characteristics of those who adhered to the DASH diet, compared with those who did not, suggested an overall healthier lifestyle. Hence, we cannot exclude residual confounding from a healthy lifestyle in domains that were not measured that would bias our results towards greater benefit of adherence. We also did not have information on the use of

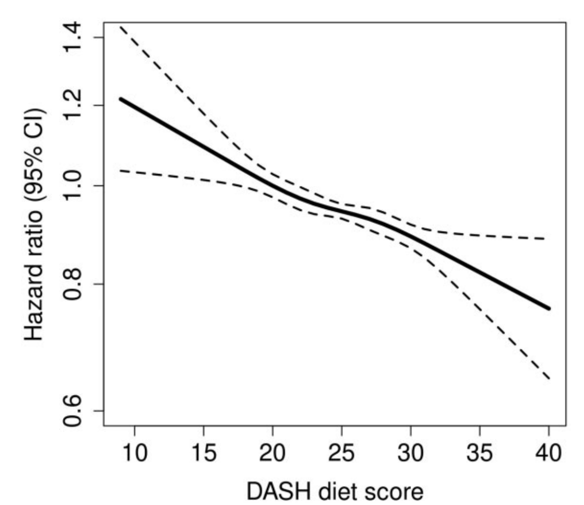


Figure 1 Association between adherence to the Dietary Approach to Stop Hypertension diet score and risk of heart failure. The Dietary Approach to Stop Hypertension diet score was modelled as a restrictive cubic spline with four knots (reference is set at 20 points). Multivariable adjusted: age, sex, education, smoking, alcohol, walking/cycling, corticosteroid use, aspirin use, dietary supplements use, use of hormone replacement therapy in women, family history of myocardial infarction and total energy intake.

cardiovascular medications beside aspirin intake and we did not have information on subtypes of HF from the register. However, in a recent study, there was not observed a difference in the association of DASH diet with incident HF with reduced ejection fraction compared with HF with preserved ejection fractions.[10]

Comparison to other studies

Only few cohort studies have looked at adherence to the DASH diet and incidence for HF and results have been conflicting. A lower risk was observed in the Multi-Ethnic Study of Atherosclerosis[9] whereas a higher risk was reported in the Cardiovascular Health Study; neither being statistically significant,[8] which could be because both cohorts included fewer cases than in our study or because the study population was older in the Cardiovascular Health Study, which has been found to modify the association in other cohorts.[10] More in line with our results, a 27% (95% CI: 8, 42%) lower risk was observed in a diverse US population.[10] The stronger association observed, compared with our study, may be due to shorter follow-up or differences in the underlying dietary patterns such as lower intake of sugar-sweetened beverages in the Swedish compared with the USA cohort. In our two previous publications, we observed that adherence to the DASH diet was associated with a lower risk of HF in men and women.[11,12] In those studies, the follow-up time was shorter and with fewer HF cases than in the present study. The point estimates were stronger in those two previous studies but had wider CIs compared to this study. The stronger point estimates may be due to the FFQs being more representative of usual diet over the shorter time period, as our sensitivity analyses with updated dietary intake and different follow-up times indicated. More broadly, adherence to the DASH has in multiple other cohorts been associated with a lower risk of cardiovascular disease and all-cause mortality.[5,28]

The initial DASH diet trial reported a 5.5-mmHg lower systolic blood pressure with the DASH diet compared with a standard American diet over 8 weeks[29] and RCTs using pharmacological interventions have demonstrated that every 5 mmHg lower systolic blood pressure lowers the risk of HF by 24%,[30] which is comparable to our estimate of 18% (95% CI: 13–23%) lower risk in the highest quintile of the DASH diet.

Potential mechanisms explaining results

Several trials of the DASH diet (0.5–12 months long) have investigated its effect on key risk markers for the development of HF. An umbrella review of meta-analyses of the RCTs concluded that consuming a DASH diet reduced blood pressure, body weight, improved glucose regulation, and lowered LDL-cholesterol compared with different control diets,[6] in line with our finding of a lower long-term risk. Also, adherence to a DASH diet lowered levels of biomarkers for cardiac strain and cardiac injury in a controlled feeding study over 4 weeks.[31]

When investigating individual food components of the DASH diet, we found that replacing red and processed meat with other DASH diet foods was associated with a lower risk. This is also consistent with results from a meta-analysis of RCTs specifying control diets to interventions of red meat; intake of red meat increased LDL-cholesterol compared with plant-protein sources such as legumes and nuts.[32] Our substitution analysis assumed a linear relationship; however, when we investigated the individual components using a flexible model, low intakes of red and processed meat (around 50 g/day) and low-fat dairy (around 200 g/day) were associated with a lower risk, whereas higher intakes were associated with a higher risk compared with no intake. This shows that red meat, an important source of haem iron, may at low intake levels prevent iron deficiency and lower risk of HF.[14] Iron deficiency is recognized in 35–55% of patients with HF, which worsens their prognosis.[14] However, higher intake, especially of processed red meat (41% of total red and processed meat consumed in our study population) containing high amounts of sodium, is related to a higher risk,[33] as suggested by a strong dose–response relation between total sodium intake and risk of HF in this study. A similar association was observed for low-fat dairy where an intake of about 150 g/day was associated with a lower HF risk, an intake comparable to the DASH diet trials, and intakes beyond this were associated with a higher risk. Very high intake of milk has been hypothesized to induce oxidative stress,[34] part of which could be due to the displacement of other foods like fruits and vegetables in the diet, and thereby be associated with a higher risk of HF. Intake of sugar-sweetened beverages has shown to increase body weight in RCTs and cohort studies,[35] and this may explain why we found a strong linear increased risk, whereas higher intake of vegetables, which has been shown to lower body weight in ad libitum settings,[36] was associated with a lower risk.

Table 3 Rate differences and 95% confidence intervals for the association between adherence to the Dietary Approach to Stop Hypertension diet score and risk of heart failure after 20 years of follow-up (n = 76 122, n cases = 10 634, person-years = 5 912 940)

	DASH diet score				
	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5
Range	8–21	22–24	25–26	27–29	30–40
Cases	2526	2580	1807	2167	1554
Person-years	1 371 560	1 364 929	977 732	1 233 664	965 055
Rate difference per 10 000 person-years (95% CI)					
Baseline diet					
Age and sex adjusted[a]	0 (ref)	−8.1 (−13.0, −3.3)	−14.3 (−19.6, −9.0)	−21.8 (−26.9, −16.7)	−30.9 (−36.3, −25.5)
Multivariable adjusted 1[b]	0 (ref)	−3.5 (−8.3, 1.39)	−6.2 (−11.3, −0.8)	−11.4 (−16.5, −6.3)	−17.3 (−22.9, −11.7)
Multivariable adjusted 2[c]	0 (ref)	−4.1 (−8.9, 0.8)	−6.9 (−12.2, −1.5)	−12.6 (−17.7, −7.5)	−18.9 (−24.5, −13.3)
Long-term diet					
Age and sex adjusted	0 (ref)	−14.1 (−19.1, −9.1)	−22.2 (−27.6, −16.8)	−25.8 (−30.9, −20.7)	−33.1 (−38.6, −27.6)
Multivariable adjusted 1	0 (ref)	−8.7 (−13.7, −3.7)	−13.8 (−19.2, −8.4)	−14.7 (−19.9, −9.5)	−19.0 (−24.7, −13.3)
Multivariable adjusted 2	0 (ref)	−9.0 (−14.0, −4.0)	−14.6 (−20.0, −9.2)	−15.2 (−20.4, −10.0)	−19.8 (−25.5, −14.1)

BMI, body mass index; CI, confidence interval; DASH, Dietary Approach to Stop Hypertension.
[a]Age and sex adjusted: age, sex.
[b]Multivariable adjusted 1: age, sex, education, smoking, alcohol, walking/cycling, corticosteroid use, aspirin use, dietary supplements use, family history of myocardial infarction.
[c]Multivariable adjusted 2: multivariable adjusted 1, BMI, history of hypertension, hypercholesterolaemia, diabetes and atrial fibrillation at baseline.

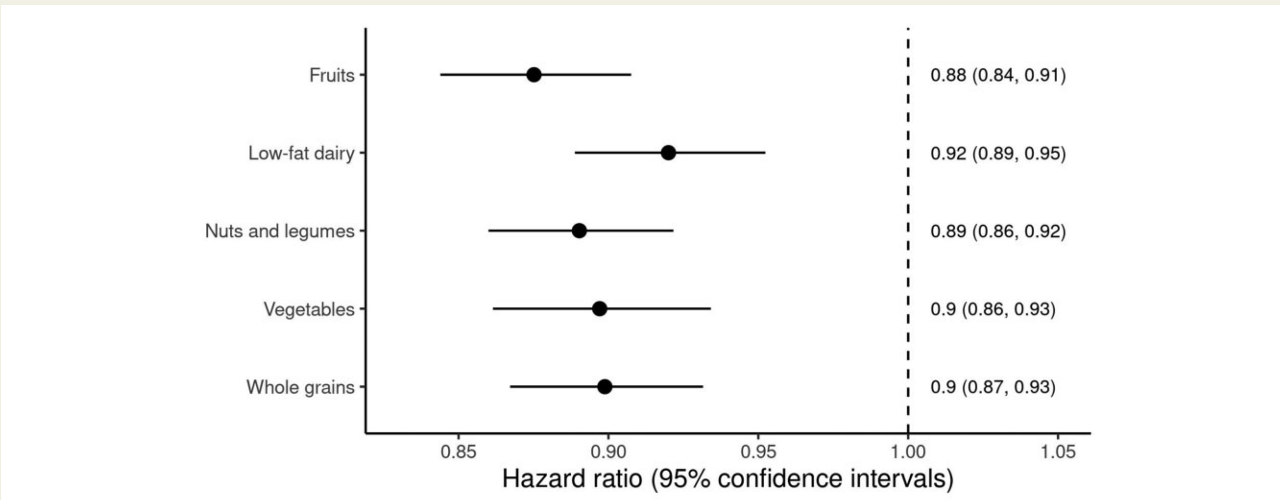


Figure 2 Replacement of red and processed meat with other Dietary Approach to Stop Hypertension diet foods on risk of heart failure. Serving sizes were 100 g/day for red and processed meat, 100 g/day for fruit, vegetables, and whole grains, 200 g/day for low-fat dairy and 20 g/day for nuts and legumes. Adjusted for each of the included foods, except for red and processed meat, a total-variable all substitution foods as well as: age, sex, education, smoking, alcohol, walking/cycling, corticosteroid use, aspirin use, dietary supplements use, use of hormone replacement therapy in women, family history of myocardial infarction and total energy intake.

Public health implications

Based on our long-term prospective study with robust findings across sensitivity analyses and findings in accordance with short-term RCTs we, under the assumption of a causal relation, estimated that between 13 and 25 cases per 10 000 person-years could have been prevented if the study population aged 45–85 years had adhered to the DASH diet. Viewed in the light of everyone being ‘exposed’, this would be an important contribution to population-based prevention efforts.

Although there are biological arguments for the investigation of effect modification by the history of diabetes or hypertension,[1] we found little evidence for effect modification, suggesting particular

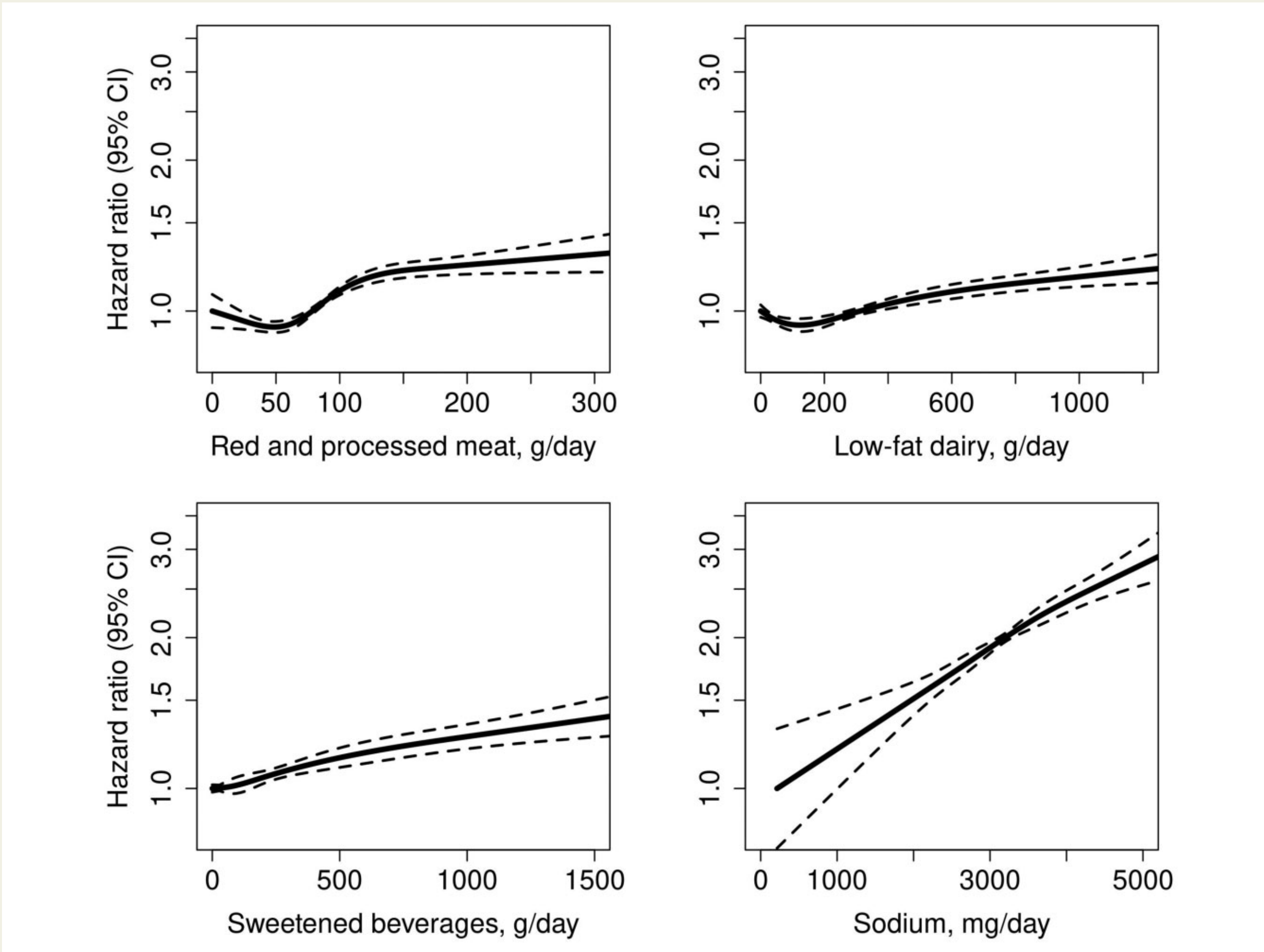


Figure 3 Association between adherence to each Dietary Approach to Stop Hypertension diet score component and risk of heart failure. Red and processed meat, low-fat dairy, sweetened beverages and sodium was modelled as restrictive cubic splines with four knots. Adjusted for: age, sex, education, smoking, alcohol, walking/cycling, corticosteroid use, aspirin use, dietary supplements use, use of hormone replacement therapy in women, family history of myocardial infarction, total energy intake and mutually adjusted for each of the other foods. Sodium was not included in models with foods.

subgroups that would benefit from adhering to the DASH diet by any of these factors on either scale. One explanation is lack of power, as these subgroups included few participants. As we used self-reported prevalence of both history of hypertension and diabetes at the baseline, measurement error could also explain why we did not observe any statistically significant differences.

Conclusion

Long-term adherence to the DASH diet was associated with a lower risk of HF in a cohort of middle-aged and elderly Swedish adults. Replacing 1 serving/day of red and processed meat with 1 serving/day of other DASH diet foods, such as fruits, vegetables, low-fat dairy, nuts and legumes, or whole grains, was also associated with about 10% lower risk of HF. Our findings, together with the evidence from relatively short RCTs, underline the importance of supporting the population in adhering to a healthy diet, like the DASH diet, for the prevention of HF in the general population.

Supplementary material

Supplementary material is available at *European Heart Journal – Cardiovascular Pharmacotherapy* online.

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Data availability

The data underlying this article cannot be shared publicly because the data are classified as sensitive data under the European General Data Protection Regulation. The data and underlying code can be shared on reasonable request to the corresponding author through the Swedish National Research Infrastructure SIMPLER (www.simpler4health.se).

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References

1. Ziaeian B, Fonarow GC. Epidemiology and aetiology of heart failure. *Nat Rev Cardiol* 2016;**13**:368–378.
2. Ewers B, Marott JL, Schnohr P, Nordestgaard BG, Marckmann P. Non-adherence to established dietary guidelines associated with increased mortality. The Copenhagen General Population Study. *Eur J Prev Cardiol* 2021;**28**:1259–1268.
3. Ibsen DB, Søgaard K, Sørensen LH, Olsen A, Tjønneland A, Overvad K, Dahm CC. Modifiable lifestyle recommendations and mortality in denmark: a cohort study. *Am J Prev Med* 2021;**60**:792–801.
4. Strengers JG, den Ruijter HM, Boer JMA, Asselbergs FW, Verschuren WMM, van der Schouw YT, Sluijs I. The association of the Mediterranean diet with heart failure risk in a Dutch population. *Nutr Metab Cardiovasc Dis* 2021;**31**:60–66.
5. Patel YR, Robbins JM, Gaziano JM, Djoussé L. Mediterranean, DASH, and Alternate Healthy Eating Index dietary patterns and risk of death in the physicians' health study. *Nutrients* 2021;**13**:1893.
6. Chiavaroli L, Viguiliouk E, Nishi S, Blanco Mejia S, Rahelić D, Kahleová H, Salas-Salvadó J, Kendall C, Sievenpiper J. DASH dietary pattern and cardiometabolic outcomes: an umbrella review of systematic reviews and meta-analyses. *Nutrients* 2019;**11**:338.
7. Patel Y, Joseph J. Sodium intake and heart failure. *Int J Mol Sci* 2020;**21**:1–12.
8. Del Gobbo LC, Kalantarian S, Imamura F, Lemaitre R, Siscovick DS, Psaty BM, Mozaffarian D. Contribution of major lifestyle risk factors for incident heart failure in older adults. The Cardiovascular Health Study. *JACC Hear Fail* 2015;**3**:520–528.
9. Campos CL, Wood A, Burke GL, Bahrami H, Bertoni AG. Dietary approaches to stop hypertension diet concordance and incident heart failure: the multi-ethnic study of atherosclerosis. *Am J Prev Med* 2019;**56**:819–826.
10. Goyal P, Balkan L, Ringel JB, Hummel SL, Sterling MR, Kim S, Arora P, Jackson EA, Brown TM, Shikany JM, Judd SE, Safford MM, Levitan EB. The Dietary Approaches to Stop Hypertension (DASH) diet pattern and incident heart failure. *J Card Fail* 2021;**27**:512–521.
11. Levitan EB, Wolk A, Mittleman MA. Consistency with the DASH diet and incidence of heart failure. *Arch Intern Med* 2009;**169**:851–857.
12. Levitan EB, Wolk A, Mittleman MA. Relation of consistency with the dietary approaches to stop hypertension diet and incidence of heart failure in men aged 45 to 79 years. *Am J Cardiol* 2009;**104**:1416–1420.
13. Ibsen DB, Laursen ASD, Würtz AML, Dahm CC, Rimm EB, Parner ET, Overvad K, Jakobsen MU. Food substitution models for nutritional epidemiology. *Am J Clin Nutr* 2021;**113**:294–303.
14. Rizzo C, Carbonara R, Ruggieri R, Passantino A, Scrutinio D. Iron deficiency: a new target for patients with heart failure. *Front Cardiovasc Med* 2021;**8**:709872.
15. Harris H, Håkansson N, Olofsson C, Stackelberg O, Julin B, Åkesson A, Wolk A. The Swedish mammography cohort and the cohort of Swedish men: study design and characteristics of 2 population-based longitudinal cohorts. *OA Epidemiol* 2013;**1**:16.
16. Messerer M, Johansson SE, Wolk A. The validity of questionnaire-based micronutrient intake estimates is increased by including dietary supplement use in Swedish men. *J Nutr* 2004;**134**:1800–1805.
17. Bergström L, Kylberg E, Hagman UE, Bruce Å. The food composition database KOST: the national food administration's information system for nutritive values of food. *Vår Föda* 1991;**43**:439–447.
18. Fung TT, Chiuve SE, McCullough ML, Rexrode KM, Logroscino G, Hu FB. Adherence to a DASH-style diet and risk of coronary heart disease and stroke in women. *Arch Intern Med* 2008;**168**:713–720.
19. Larsson SC, Håkansson N, Näslund I, Bergkvist L, Wolk A. Fruit and vegetable consumption in relation to pancreatic cancer risk: a prospective study. *Cancer Epidemiol Biomarkers Prev* 2006;**15**:301–305.
20. Larsson SC, Giovannucci E, Bergkvist L, Wolk A. Whole grain consumption and risk of colorectal cancer: a population-based cohort of 60 000 women. *Br J Cancer* 2005;**92**:1803–1807.
21. Larsson SC, Bergkvist L, Wolk A. Milk and lactose intakes and ovarian cancer risk in the Swedish Mammography Cohort. *Am J Clin Nutr* 2004;**80**:1353–1357.
22. Genkinger JM, Friberg E, Goldbohm RA, Wolk A. Long-term dietary heme iron and red meat intake in relation to endometrial cancer risk. *Am J Clin Nutr* 2012;**96**:848–854.
23. Larsson SC, Bergkvist L, Wolk A. Consumption of sugar and sugar-sweetened foods and the risk of pancreatic cancer in a prospective study. *Am J Clin Nutr* 2006;**84**:1171–1176.
24. Ingelsson E, Ärnlöv J, Sundström J, Lind L. The validity of a diagnosis of heart failure in a hospital discharge register. *Eur J Heart Fail* 2005;**7**:787–791.
25. Rod NH, Lange T, Andersen I, Marott JL, Diderichsen F. Additive interaction in survival analysis: use of the additive hazards model. *Epidemiology* 2012;**23**:733–737.
26. Yeh ETH, Bickford CL. Cardiovascular complications of cancer therapy: incidence, pathogenesis, diagnosis, and management. *J Am Coll Cardiol* 2009;**53**:2231–2247.
27. Hernán MA. The hazards of hazard ratios. *Epidemiology* 2010;**21**:13–15.
28. Schwingshackl L, Hoffmann G. Diet quality as assessed by the healthy eating index, the alternate healthy eating index, the dietary approaches to stop hypertension score, and health outcomes: a systematic review and meta-analysis of cohort studies. *J Acad Nutr Diet* 2015;**115**:780–800.e5.
29. Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, Bray GA, Vogt TM, Cutler JA, Windhauser MM, Lin P-H, Karanja N, Simons-Morton D, McCullough M, Swain J, Steele P, Evans MA, Miller ER, Harsha DW. A clinical trial of the effects of dietary patterns on blood pressure. *N Engl J Med* 1997;**336**:1117–1124.
30. Danelich I, Reed B, Sueta C. Stage A: can heart failure be prevented? *Curr Cardiol Rev* 2014;**11**:4–9.
31. Juraschek SP, Kovell LC, Appel LJ, Miller ER, Sacks FM, Chang AR, Christenson RH, Rebuck H, Mukamal KJ. Effects of diet and sodium reduction on cardiac injury, strain, and inflammation. *J Am Coll Cardiol* 2021;**77**:2625–2634.
32. Guasch-Ferré M, Satija A, Blondin SA, Janiszewski M, Emlen E, O'Connor LE, Campbell WW, Hu FB, Willett WC, Stampfer MJ. Meta-analysis of randomized controlled trials of red meat consumption in comparison with various comparison diets on cardiovascular risk factors. *Circulation* 2019;**139**:1828–1845.
33. Kaluza J, Åkesson A, Wolk A. Long-term processed and unprocessed red meat consumption and risk of heart failure: a prospective cohort study of women. *Int J Cardiol* 2015;**193**:42–46.
34. Michaelsson K, Wolk A, Langenskiold S, Basu S, Warensjo Lemming E, Melhus H, Byberg L. Milk intake and risk of mortality and fractures in women and men: cohort studies. *BMJ* 2014;**349**:g6015–15.
35. Malik VS, Hu FB. Sugar-sweetened beverages and cardiometabolic health: an update of the evidence. *Nutrients* 2019;**11**:1840–1817.
36. Mytton OT, Nnoaham K, Eyles H, Scarborough P, Ni Mhurchu C. Systematic review and meta-analysis of the effect of increased vegetable and fruit consumption on body weight and energy intake. *BMC Public Health* 2014;**14**:886.