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The Dietary Approaches to Stop Hypertension (DASH) Diet Pattern and Incident Heart Failure

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Abstract

Background: The Dietary Approaches to Stop Hypertension (DASH) diet pattern has shown some promise for preventing heart failure (HF), but studies have been conflicting.

Objective: To determine whether the DASH diet pattern was associated with incident HF in a large biracial and geographically diverse population.

Methods: Among participants in the REasons for Geographic And Racial Differences in Stroke (REGARDS) cohort study of adults aged ≥45 years who were free of suspected HF at baseline in 2003–2007, DASH diet score was derived from the baseline food frequency questionnaire. The main outcome was incident HF defined as the first adjudicated HF hospitalization or HF death through December 31, 2016. We estimated hazard ratios (HR) for the associations of DASH diet score quartiles with incident HF, and incident HF with reduced ejection fraction (HFrEF) and preserved ejection fraction (HFpEF) using the Lunn-McNeil extension to the Cox model. We tested for several pre-specified interactions including with age.

Results: Compared to the lowest quartile, individuals in the 2nd-4th DASH diet score quartiles had lower risk for incident HF after adjustment for sociodemographic and health characteristics:

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Q2 HR 0.70, 95%CI[0.56–0.87]; Q3 HR 0.65, 95%CI:[0.52–0.81]; Q4 HR 0.67, 95%CI:[0.52–0.85]. When stratifying results by age, quartiles 2–4 had a lower hazard for incident HF among age<65 years, quartiles 3–4 had a lower hazard among age 65–74, and the quartiles had similar hazard among age ≥75 years (p-for-interaction=0.003). We did not find a difference in the association of DASH diet with incident HFrEF vs. HFpEF (p=0.11).

Conclusions: DASH diet adherence was inversely associated with incident HF, specifically among individuals <75 years old.

Keywords

heart failure; incident heart failure; DASH diet

INTRODUCTION:

Heart failure (HF) affects over 6 million people across the United States(1), and is responsible for a significant portion of healthcare costs, as well as considerable morbidity and mortality. Unfortunately, despite significant advances in HF care over the past 2 decades, outcomes for adults with HF remain poor, with a 5-year mortality above 50% irrespective of HF subtype.(2) Accordingly, the best strategy to improve outcomes is likely to prevent the development of HF.(3)

Behavior and lifestyle represent the cornerstones of primary prevention for many cardiovascular conditions including HF. Over the past several years, there has been increased attention on identifying diet patterns that may be protective against HF. A recent study from the REasons for Geographic And Racial Differences Study (REGARDS) cohort examining diet patterns derived from a factor analysis showed that a plant-based diet pattern was inversely associated with incident HF, while a Southern diet pattern was positively associated with incident HF.(4) Because hypertension is one of the most important modifiable risk factors for HF, diet patterns that reduce blood pressure are particularly appealing for the prevention of HF. The well-known Dietary Approaches to Stop Hypertension (DASH) diet was designed as a palatable, accessible style of eating for US adults that is low in sodium and high in potassium, rich in fruits and vegetables, moderate in low-fat dairy products, and low in red and processed meats, with a substantial amount of plant protein from legumes and nuts. Randomized controlled trials have shown that the DASH diet lowers blood pressure(5) and reduces low-density lipoprotein (LDL) cholesterol.(6) The DASH diet is also associated with favorable changes in left ventricular systolic parameters(7) and diastolic function.(8) While it shares some features with the plant-based diet pattern derived among REGARDS participants, the DASH diet pattern is distinct from the plant-based diet pattern in that it highlights several specific features, including low intake of red and processed meats, sodium, and sugar-sweetened beverages.

Prior studies examining the association of a DASH diet and HF have had conflicting results. Two studies from Sweden showed that the DASH diet was inversely associated with incident HF among women(9) and men.(10) In contrast to these studies, findings from the Cardiovascular Health Study showed no association between DASH diet and incident HF.(11) Importantly, these studies had limited generalizability, as they each examined

cohorts predominantly comprised of white participants. Moreover, none of these studies examined specific subtypes of HF, which may be important given key differences in the pathophysiologies of HF with preserved ejection fraction (HFpEF) and HF with reduced ejection fraction (HFrEF).(12) Thus, we examined the association between the DASH diet pattern and HF among participants in the REGARDS cohort, which is particularly well-suited for this inquiry because it is a geographically-diverse population that includes men and women, a broad range of ages, and a significant number of blacks (in addition to whites), and provides detail on HF subtypes (HFpEF and HFrEF).

METHODS:

Data Source

The study population was derived from the REGARDS cohort, which has been previously described.(13) Briefly, the REGARDS cohort is comprised of 30,239 community dwelling participants (42% blacks and 55% women) residing in the continental United States. Participants were recruited from 2003 to 2007 with ongoing follow-up. The cohort was initially designed to address geographic and racial differences in stroke mortality and therefore over-selected for populations with higher risk for stroke including black adults and those residing in the southeastern United States in areas known as the stroke belt (an area in the Southeastern U.S. with high stroke mortality that includes North Carolina, South Carolina, Georgia, Alabama, Mississippi, Tennessee, Arkansas, and Louisiana) and stroke buckle (an area with even higher stroke mortality that includes the coastal plains of North Carolina, South Carolina, and Georgia).

Baseline data collection for REGARDS was completed using computer-assisted telephone interviews to collect medical history, functional status, health behaviors, and psychosocial measures. In-home examinations were conducted by trained health care professionals using standardized, quality-controlled protocols to collect physiologic and anthropometric measures (blood pressure, height, weight, waist circumference), blood and urine samples, electrocardiograms, and medication use by pill bottle review. Food frequency questionnaires were completed by participants following the in-home visit. Blood and urine samples were centrally analyzed at the University of Vermont.

The REGARDS study was previously approved by the Institutional Review Boards at the participating centers. This ancillary study was approved by the Weill Cornell Institutional Review Board. All participants provided written informed consent.

Study Population

We studied participants without suspected baseline HF from the REGARDS cohort, who had completed food frequency questionnaires. This HF-free cohort was created based on the use of HF-specific medication, and has a negative predictive value of >95% compared to diagnoses in Medicare claims (14). Among 25,790 participants in the HF-free cohort, we excluded those with missing or incomplete (<85% completion) food frequency questionnaires and participants with biologically implausible energy intake (men with an intake of <3347 kJ/d or >20,920 kJ/d; and women with an intake of <2093 kJ/d or >18,841

kJ/d), as has been done in prior studies.(15) (Figure 1). Supplemental Table 1 shows differences between participants with complete dietary data (n=18,856) and those with missing dietary data (n=6934).

Primary Outcome:

The primary outcome was an adjudicated incident HF event, defined as an adjudicated HF hospitalization or HF death through December 31, 2016; adjudication was performed independently by 2 clinician investigators with disagreements resolved by discussion. Adjudication of a HF hospitalization was based on signs and symptoms, laboratory studies, electrocardiogram, and assessments of left ventricular function documented in the medical records.(16) Adjudication of a death with HF as the underlying cause was based on interviews with participant proxies, medical records for hospitalizations around the time of death, and death certificates. HF-subtypes were identified based on left ventricular ejection fraction (EF) obtained at the time of an incident event—HFrEF was defined as EF <50% or qualitative report of reduced EF; and HFpEF was defined as EF ≥50% or qualitative report of preserved EF.(17) Subtyping HF was not possible for incident events based on a HF-related death.

Key Explanatory Variable:

The primary exposure variable was DASH diet score. The DASH diet score was calculated as the sum of 8 component scores based on dietary intake data from the Block 98 Food Frequency Questionnaire across the following domains: (1) fruits, (2) vegetables, (3) nuts and legumes, (4) low-fat dairy products, (5) whole grains, (6) sodium, (7) sweetened beverages, and (8) red and processed meats.(18) Component scores for each domain were determined based on population-specific quintiles. For components 1 through 5, participants received a score of 5 if they were in the highest quintile and a score of 1 if they were in the lowest quintile. For components 6–8, participants received a score of 1 if they were in the highest quintile and a score of 5 if they were in the lowest quintile. The final DASH diet score ranged from 8–40, where 8 indicated the lowest adherence to the DASH diet and 40 indicated highest adherence to the DASH diet. We categorized each participant into a quartile based on their DASH diet score.

Covariates:

We chose covariates for our statistical models a priori based on the literature and clinical expertise. These variables included baseline total energy intake, sociodemographic characteristics, health behaviors, physiologic parameters, and cardiovascular comorbid conditions (diabetes, coronary heart disease, and atrial fibrillation). Given their association with incident HF, similar variables have been used in prior studies of incident HF using REGARDS.(16,19)

Statistical Analysis

We calculated descriptive statistics for baseline participant characteristics (mean with standard deviation [SD] for continuous variables or percentages for categorical variables).

We then calculated the incident rates of HF, HFpEF, and HFrEF for each DASH score quartile as number of cases per 10,000-person years.

We used Cox proportional-hazards regression models to determine whether the DASH diet score was associated with incident HF events. Model 1 included total energy intake and socio-demographic factors (age, sex, and race); Model 2 included Model 1 plus annual family income, educational achievement, insurance type, marital status, geographic region, cigarette smoking, alcohol consumption (none, moderate [0–7 drinks per week for women, 0–14 drinks per week for men], or heavy [>7 drinks per week for women, >14 drinks for per week for men], defined according to the National Institute on Alcohol Abuse and Alcoholism), and physical activity (based on amount of exercise per week); and Model 3 included Model 2 plus potentially mediating physiologic parameters and comorbid conditions including systolic blood pressure, diastolic blood pressure, body mass index, high-density lipoprotein cholesterol, total cholesterol, triglycerides, estimated glomerular filtration rate, natural log-transformed C-reactive protein, urine albumin to creatinine ratio, left ventricular hypertrophy, diabetes, coronary heart disease, and atrial fibrillation.

A global test of violation of the proportion hazards assumption using Schoenfeld residuals was significant and individual tests of the proportional hazards assumption indicated that the associations of diabetes and age varied over follow-up. Therefore, in the adjusted models we allowed the baseline hazard to vary by age and diabetes.

To account for missing covariate values in regression analysis, we applied multiple imputation using the Markov Chain Monte Carlo algorithm designed for single-level and multi-level data.(20) The variables with the greatest percent missing were income (11.5%) and CRP (5.5%).

To ensure the robustness of our findings, we conducted several sensitivity analyses. Given the competing risk of mortality, we conducted a sensitivity analysis to examine the association between DASH diet score and incident HF using Fine and Gray competing risk models. For improved granularity regarding a potential threshold for an association between DASH diet score and incident HF, we also examined the association between DASH diet score and incident HF using DASH diet deciles instead of quartiles.

Given the potential influence of baseline coronary heart disease, hypertension (according to baseline measured systolic blood pressure [SBP]), and/or chronic kidney disease (according to baseline estimated glomerular filtration rate [eGFR]) on an association between DASH diet and incident HF, we also examined for possible interactions with these comorbid conditions by adding cross-product terms to the models and performing a Wald test. Given known sex and race-based differences in left ventricular remodeling in response to stressors, we also examined whether associations between the DASH diet score and incident HF differed according to sex and race. Finally, given the possibility that the benefits of the DASH diet might attenuate with increasing age, we examined whether associations between the DASH diet score and incident HF differed according to age <65 years, 65–74 years, or ≥75 years. For interactions where the $p<0.10$, we presented stratum-specific analyses.

Given well-described differences in the demographics, pathophysiology, treatment, and disease trajectory of HFrEF compared to HFpEF, we repeated the regression models to determine whether the DASH diet score was differentially associated with incident HFrEF and HFpEF using a Lunn-McNeil extension to the Cox proportional hazards model.(21)

We used two-sided hypothesis testing, and p-value <0.05 to determine statistical significance for main effects and p-value <0.10 to determine statistical significance of interaction terms. We managed the data in SAS version 9.4 (SAS Institute, Cary, NC) and performed statistical analysis using STATA version 14 (IBM corporation, Armonk, NY).

RESULTS:

Study Population

The mean age of participants was 64.0 (SD 9.2) years, 55.9% were female, and 32.5% were black (Table 1). The median DASH diet score was 24 with an interquartile range of 21–27. When compared to participants in the lowest DASH diet quartile, participants in DASH diet quartiles 2 through 4 were older, more frequently white, more likely to have completed high school, and had higher household incomes. Non-smokers and individuals with moderate alcohol use and more frequent exercise were more common among the higher DASH diet quartiles. Diabetes mellitus was more common among the higher DASH quartiles while hypertension and left ventricular hypertrophy were more common in the lowest DASH diet quartile. Intake of selected nutrients stratified by DASH diet quartile are shown in Supplemental Table 2. Notably, mean sodium intake was similar across quartiles, with a narrow range of 2212–2313 mg/d.

Association Between DASH Diet and HF Events

Among this cohort, there were 767 incident HF events over a median of 10.1 years of follow-up, for an incidence rate of 44/10,000 person-years. Among the 767 events, 656 were HF hospitalizations and 111 were HF-related deaths.

The incidence of HF was highest among those in the lowest DASH diet quartile (Table 2 and Figure 2A). Participants in quartiles 2–4 had a statistically significantly lower hazard for incident HF compared to the lowest quartile of DASH diet score in the fully adjusted model. These results were largely unchanged when using a Fine and Gray competing risk model to account for mortality (Supplemental Table 3).

When examining the association of DASH diet score deciles and incident HF, we found that the 3rd through 10th decile (DASH score > 20) were associated with a significantly lower incident HF compared to the 1st decile (Supplemental Table 4).

We did not find evidence to support interaction of the DASH diet score with baseline coronary heart disease (p-for-interaction=0.38), baseline hypertension (baseline SBP≥130: p-for-interaction=0.49; SBP≥140: p-for-interaction=0.63), baseline chronic kidney disease (baseline eGFR<60: p-for-interaction=0.93; eGFR<45: p-for-interaction=0.15), sex (p-for-interaction= 0.41), or race (p-for-interaction=0.54). The p-for-interaction for age was 0.003. Participants in lower DASH diet score quartiles had a higher risk for developing incident

HF among individuals aged <65 years (Figure 2B) and aged 65–74 years (Figure 2C); those aged ≥75 years had similar risk of HF irrespective of DASH diet quartile (Figure 2D). In a fully-adjusted model, participants in quartiles 2–4 had a lower hazard for incident HF compared to the lowest quartile of DASH diet score among those aged <65 years, participants in quartiles 3–4 had a lower hazard for incident HF compared to the lowest quartile of DASH diet score among those aged 65–74 years; and the quartiles had similar hazard for incident HF among those aged ≥75 years old (Table 2).

Among 767 incident events, 574 had data on EF at the time of the event. There were 321 incident cases of HFrEF and 253 cases of incident HFpEF. Using a Lunn-McNeil model, we did not find a statistical difference in the association of DASH diet quartiles with incident HFrEF compared to incident HFpEF (p=0.11). Supplemental Table 5 shows hazard ratios for HFrEF and HFpEF separately.

DISCUSSION:

In this real-world, geographically diverse cohort of 18,856 participants, we found that the DASH diet was inversely associated with incident HF, specifically among those younger than 75 years. Given the ongoing HF epidemic, there is a need to identify population-based strategies to curb the incidence of HF. While control of traditional risk factors including hypertension, diabetes, and coronary artery disease has been beneficial, all cases of HF may not be fully explained by these traditional risk factors.(22) Thus, there may be value in identifying risk factors that are upstream from traditional risk factors which can subsequently be targeted. Diet has been identified as an important component of primary prevention for other cardiovascular conditions like hypertension and ischemic coronary disease.(15,18,23) Our study shows that, even when controlling for these other precipitating conditions and contributing factors like exercise, the DASH diet was associated with lower risk of incident HF. Moreover, these findings suggest that the benefit does not require extremely high DASH scores, and that avoiding low DASH scores is sufficient to reduce risk. Several other diets such as a plant-based diet and a Mediterranean diet have previously been studied. A recent retrospective study demonstrated that the highest quartile of following a plant-based diet was inversely associated with incident HF(4); and randomized controlled trials have shown that the Mediterranean diet may prevent incident HF but those studies were underpowered to make definitive conclusions(24). Given the potential benefit of these other diets which have some overlapping features with the DASH diet, future studies examining the interplay between specific macro or micronutrient components of these diets and incident HF may be helpful to better understand the role of diet in preventing HF.

Prior work in this area has been limited to select populations and has consequently revealed conflicting results. In Sweden, DASH diet correlated with a 37% lower incidence of HF in women(9) and 22% lower incidence in men(10) comparing the top to bottom quartiles. However, these studies examined a homogenous white population. A study of 4,490 individuals from the Cardiovascular Health Study showed no benefit; importantly, this study included an older population (mean age 72 years) that was again predominantly (89%) white. A recent study of 4,478 participants from Multi-Ethnic Study of Atherosclerosis (25)

showed lower incident HF events in those less than 75 years old, but there were only 179 total events, yielding wide confidence intervals.(25) In contrast to prior studies, our study examined a geographically diverse United States population, which notably included 32.5% blacks and an age range of 45–98 years. Accordingly, our study helps to reconcile some of the conflicting findings of prior studies. In particular, we found that the DASH diet was potentially protective among those younger than 75 years, but not in individuals 75 years or older. This extends findings from the prior Multi-Ethnic Study of Atherosclerosis study and may help explain the differences in findings between the Swedish cohorts and the Cardiovascular Health Study (mean ages 61 years in the Swedish women and 59 years in the Swedish men, compared to 72 years in the Cardiovascular Health Study).(11,25) These findings suggest that the relative importance of diet may be less important with advancing age, perhaps due to the accumulation of other risk factors which may supersede the benefits of diet. Future research is needed to confirm the interaction between DASH diet and age and to elucidate potential mechanisms. Our study also showed that the DASH diet was similarly protective in both men and women, and in both white and black participants. We had hypothesized that the DASH diet would demonstrate a stronger effect in black persons given prior data demonstrating that DASH had an especially potent effect on blood pressure in blacks persons compared to white persons.(26) Our observation here of a similar association in both black and white persons suggests that the mechanism of protection may extend beyond blood pressure control. Taken together, our findings support the importance of implementing strategies for primary prevention earlier in the lifespan, irrespective of other demographic factors like sex or race.

This is the first study to our knowledge that examined the association of the DASH diet pattern with HF subtypes. The potential protective nature of a DASH diet appeared stronger for HFrEF than HFpEF, though the difference was not statistically significant. This was not surprising given the effect of the DASH diet on several shared pathophysiologic mechanisms of HFrEF and HFpEF. Indeed, the DASH diet can decrease blood pressure (5,27), LDL cholesterol,(6) serum inflammatory biomarkers,(28,29) and activation of the renin-angiotensin system,(30,31), and improve vascular and endothelial function(32)—factors that have been implicated in the pathophysiology of HFrEF and/or HFpEF. Relatedly, the DASH diet has also been associated with better left ventricular parameters including systolic and diastolic function.(33) Thus, our findings now reinforce a biologically plausible link between the DASH diet and both incident HFrEF and incident HFpEF independent of hypertension. Whether gut microbiota, which is an emerging area of inquiry in HF(34), mediates these associations is unclear and merits further investigation.

Strengths and Limitations:

There were several strengths of this study including the large sample size, geographic and racial diversity, and adjudicated HF outcomes which were subtyped into HFrEF and HFpEF. We also incorporated a broad array of covariates into the statistical models which controlled for several domains including socio-demographics, healthy behaviors, and physiologic parameters; and conducted several sensitivity analyses to maximize the robustness of findings. There were also several important limitations that merit attention. First, diet was self-reported using a food-frequency questionnaire, an approach with known limitations.(35)

We also only included participants with at least 85% completed dietary data, which is a somewhat selected population. For example, a significant number of black participants were excluded due to missing dietary data. Although we did not observe an interaction with race, missing data could have influenced our findings. Second, the food frequency questionnaire was completed at a single time point, and may not reflect DASH diet adherence over the course of years. This concern is somewhat mitigated by the observation that dietary patterns are often stable over time.(32) Third, we lacked detailed information on cardiac function at baseline and other potential confounders and mediators. For example, we did not have data on ventricular function and used a proxy for HF diagnosis prior to enrollment. Although we adjusted for multiple confounders, there was the risk of residual confounding. Models adjusted for potential mediators through which the DASH diet may influence HF risk did not show substantial attenuation of the association. This may be because habitual diet patterns influence life-long risk factor burden, and this study included a single time point measurement of blood pressure and other HF risk factors. There are likely other mediators contributing to our finding that we did not capture—for example, our multivariable models did not include intervening cardiac events such as myocardial infarction or incident atrial fibrillation which could have played a role in the development of HF. Future work to better understand mediators of the association observed here between DASH diet and incident HF may be warranted. HF events were adjudicated based on hospitalizations and death, and thus did not capture incident HF diagnosed during an ambulatory visit. Although about a third of incident HF may be diagnosed in the ambulatory setting, a substantial proportion of individuals diagnosed with HF in the ambulatory setting are hospitalized within a year of diagnosis and would have thus been captured as an incident HF event at that time.(36) Finally, we observed a total of 574 events that were categorized as either HFrEF (n=321) or HFpEF (n=253), which limited power to detect subtle differences in the association of DASH diet with these HF subtypes.

CONCLUSIONS:

Adherence to the DASH diet pattern was associated with a reduced risk of incident HF for adults aged less than 75 years old, supporting its use for primary prevention of HF at a younger age.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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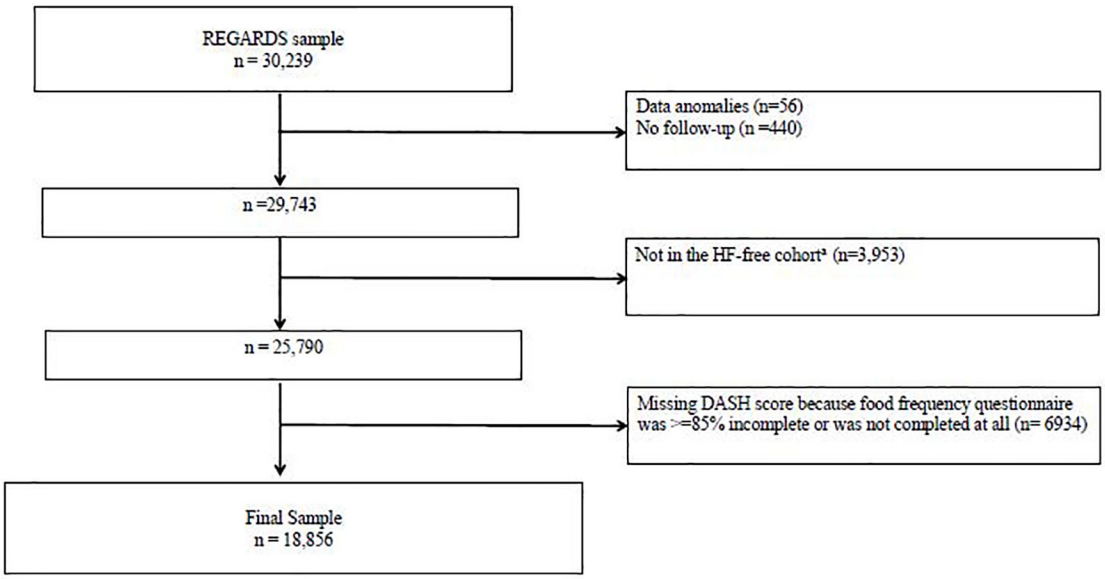
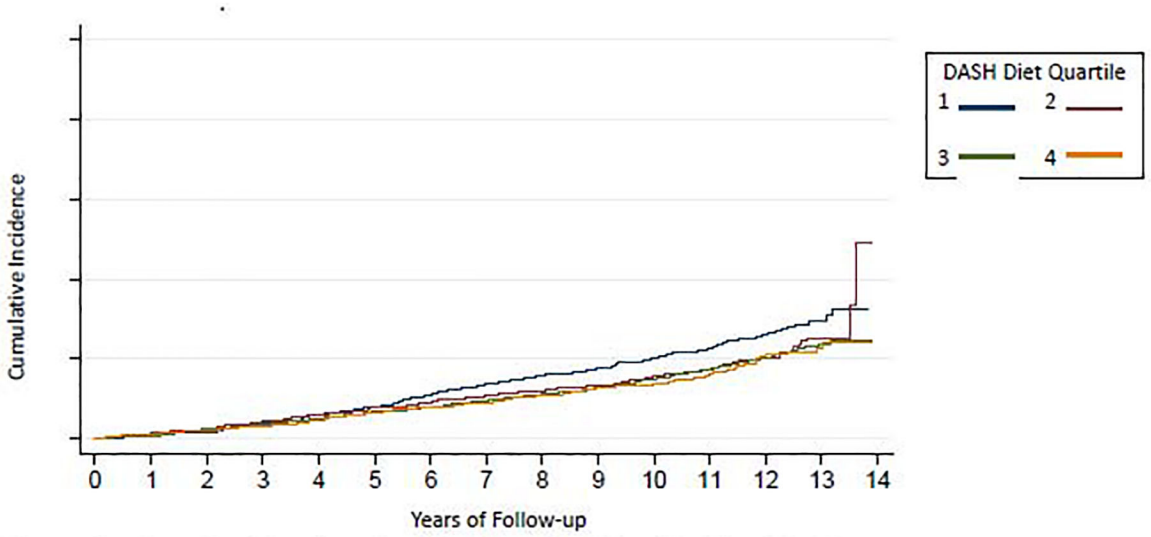


Figure 1:

Inclusion/Exclusion Cascade

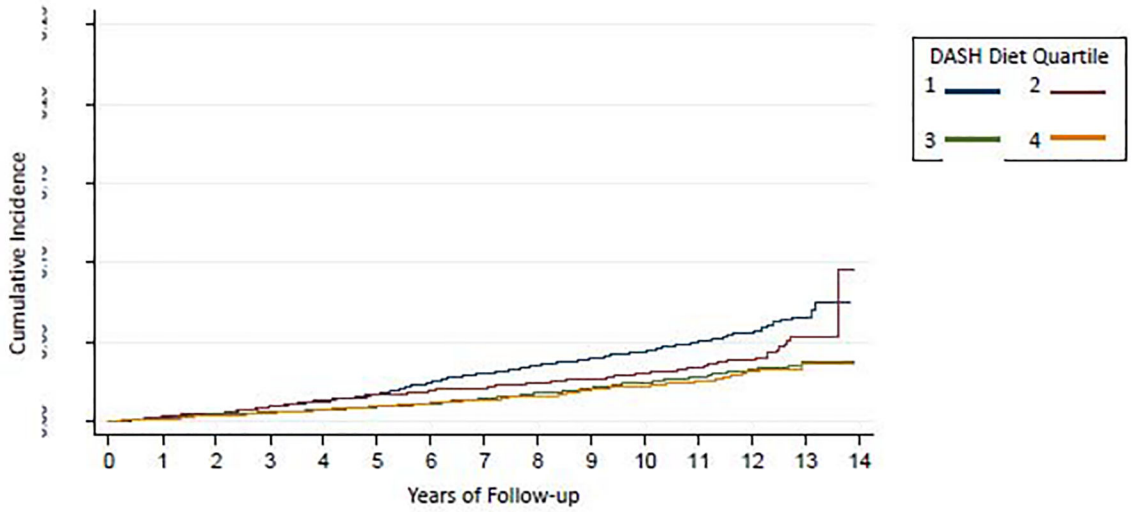
Abbreviations: REGARDS- Reasons for geographic and racial differences in strokes; HF-heart failure

A



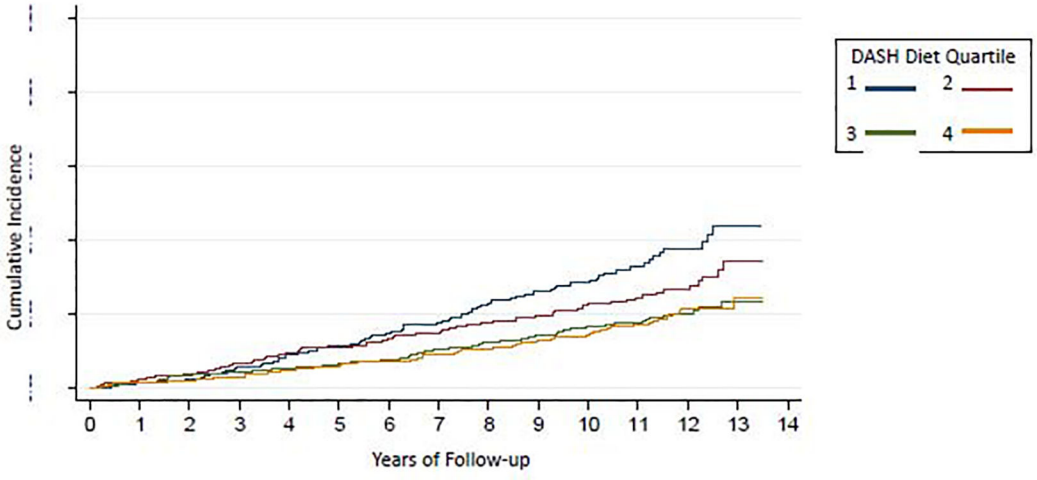
Quartile	0	1	2	3	4	5	6	7	8	9	10	11	12	13
Q1(8-20)	4203	4104	3960	3777	3586	3405	3188	3012	2818	2598	2059	1518	965	323
Q2(21-23)	4559	4463	4325	4181	3982	3786	3594	3422	3207	3006	2385	1769	1057	360
Q3(24-27)	5764	5656	5519	5339	5149	4937	4721	4516	4281	4050	3218	2406	1455	486
Q4(28-40)	4330	4260	4161	4049	3903	3762	3595	3459	3293	3106	2467	1854	1074	332

B



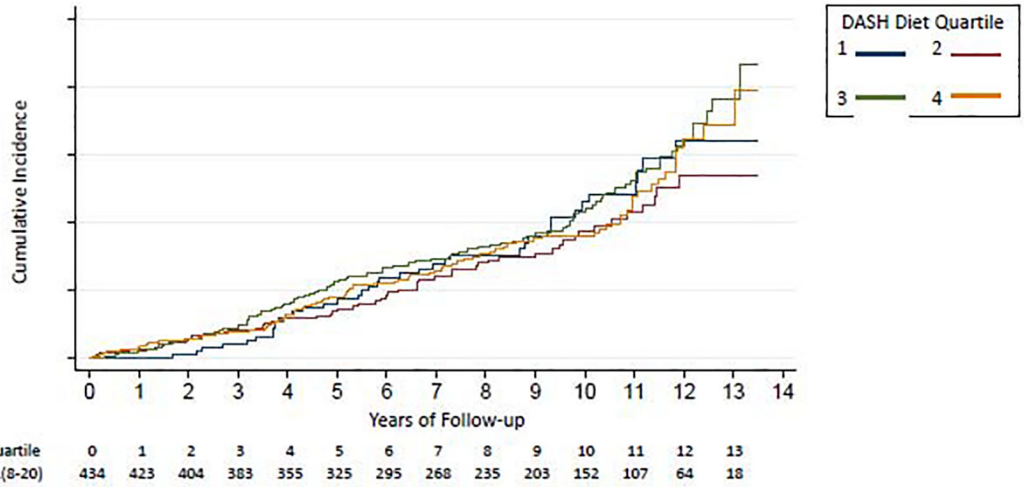
Quartile	0	1	2	3	4	5	6	7	8	9	10	11	12	13
Q1(8-20)	2624	2558	2466	2359	2234	2140	2005	1902	1804	1690	1332	985	633	221
Q2(21-23)	2427	2379	2308	2228	2141	2049	1973	1899	1805	1720	1358	997	596	210
Q3(24-27)	2865	2812	2756	2669	2594	2506	2406	2323	2231	2139	1697	1248	744	241
Q4(28-40)	1927	1905	1864	1823	1772	1715	1653	1616	1561	1487	1180	878	492	166

C



Quartile	0	1	2	3	4	5	6	7	8	9	10	11	12	13
Q1(8-20)	1145	1123	1090	1035	997	940	888	842	779	705	575	426	268	84
Q2(21-23)	1457	1429	1385	1339	1268	1210	1141	1089	1012	942	777	596	371	126
Q3(24-27)	1941	1909	1858	1803	1745	1672	1608	1536	1454	1381	1112	869	543	195
Q4(28-40)	1579	1552	1514	1478	1417	1379	1322	1264	1215	1150	941	735	458	133

D



Quartile	0	1	2	3	4	5	6	7	8	9	10	11	12	13
Q1(8-20)	434	423	404	383	355	325	295	268	235	203	152	107	64	18
Q2(21-23)	675	655	632	614	573	527	480	434	390	344	250	176	90	24
Q3(24-27)	958	935	905	867	810	759	707	657	596	530	409	289	168	50
Q4(28-40)	824	803	783	748	714	668	620	579	517	469	346	241	124	33

Figure 2:

Kaplan-Meier Curve for Association of DASH Diet quartiles and incident HF

A. All

B. Age <65 years

C. Age 65–74 years

D. Age ≥75 years

Table 1.

Participant characteristics according to DASH Diet Score Quartiles

Characteristic	Total	Q1(≥ 20)	Q2(21–23)	Q3(24–27)
N	18856	4203	4559	5764
Age, mean (SD[#])	64 (9.2)	62 (9)	64 (9.2)	65 (9.1)
Female	10535 (55.9%)	2348 (55.9%)	2474 (54.3%)	3271 (56.7%)
Black	6130 (32.5%)	1804 (42.9%)	1619 (35.5%)	1716 (29.8%)
Region				
Nonbelt	8313 (44.1%)	1767 (42.0%)	1953 (42.8%)	2571 (44.6%)
Belt	6452 (34.2%)	1526 (36.3%)	1615 (35.4%)	1882 (32.7%)
Buckle	4091 (21.7%)	910 (21.7%)	991 (21.7%)	1311 (22.7%)
Married	11797 (62.6%)	2439 (58.0%)	2820 (61.9%)	3676 (63.8%)
Less than high school education	1657 (8.8%)	500 (11.9%)	498 (10.9%)	426 (7.4%)
Annual income Less than $20k	2754 (16.5%)	835 (22.1%)	752 (18.6%)	761 (15.0%)
No Health Insurance	1180 (6.3%)	406 (9.7%)	332 (7.3%)	283 (4.9%)
Smoking status				
Current	2618 (13.9%)	1087 (26.0%)	768 (16.9%)	547 (9.5%)
Past	7585 (40.4%)	1543 (36.9%)	1808 (39.8%)	2420 (42.1%)
Never	8585 (45.7%)	1555 (37.2%)	1966 (43.3%)	2783 (48.4%)
Alcohol use				
Heavy	859 (4.6%)	229 (5.6%)	211 (4.7%)	261 (4.6%)
Moderate	6852 (37.0%)	1371 (33.3%)	1581 (35.3%)	2223 (39.2%)
None	10825 (58.4%)	2519 (61.2%)	2686 (60.0%)	3194 (56.3%)
Times per week of exercise				
4 or more per week	5785 (31.1%)	1012 (24.4%)	1240 (27.6%)	1820 (32.0%)
1 to 3 time per week	7039 (37.8%)	1441 (34.7%)	1674 (37.3%)	2268 (39.9%)
None	5789 (31.1%)	1700 (40.9%)	1571 (35.0%)	1602 (28.2%)
Body mass index (kg/m2), mean (SD)	29 (5.8)	29 (6.2)	29 (5.9)	29 (5.7)
Waist circumference (cm), mean (SD)	95 (15)	96 (15)	96 (15)	94 (15)
Calories, median (IQR)	1586 (1199, 2084)	1589 (1187, 2118)	1576 (1166, 2091)	1570 (1188, 2080)
Diabetes	2990 (16.4%)	606 (14.9%)	749 (17.0%)	967 (17.3%)
Hypertension	10349 (54.9%)	2413 (57.4%)	2603 (57.1%)	3099 (53.8%)
SBP[?] (mmHg), mean (SD)	126 (16)	128 (16)	127 (16)	126 (16)
DBP[†] (mmHg), mean (SD)	76 (9.4)	77 (9.7)	77 (9.5)	76 (9.1)
Atrial Fibrillation	1386 (7.4%)	306 (7.3%)	320 (7.0%)	422 (7.3%)
History of Ischemic Heart Disease	2647 (14.1%)	548 (13.1%)	643 (14.2%)	809 (14.1%)
Left Ventricular Hypertrophy	1548 (8.3%)	366 (8.7%)	404 (8.9%)	473 (8.3%)
Dyslipidemia	10588 (58.0%)	2347 (57.7%)	2628 (59.6%)	3258 (58.4%)
Total Cholesterol (mg/dL), mean (SD)	194 (39)	196 (40)	195 (40)	193 (39)

Characteristic	Total	Q1(≥ 20)	Q2(21–23)	Q3(24–27)
HDL[§] (mg/dL), mean (SD)	53 (16)	51 (16)	52 (16)	53 (17)
Triglycerides (mg/dL), median (IQR)	110 (81, 157)	114 (84, 163)	114 (84, 162)	109 (80, 157)
eGFR[ǂ], mean (SD)	86 (18)	88 (20)	86 (19)	85 (18)
Urinary ACR[*] (mg/g)				
ACR<10	11886 (65.7%)	2617 (65.3%)	2817 (64.2%)	3670 (66.0%)
ACR 10–29	4036 (22.3%)	849 (21.2%)	1035 (23.6%)	1254 (22.6%)
ACR 30–300	1861 (10.3%)	459 (11.4%)	448 (10.2%)	547 (9.8%)
ACR > 300	312 (1.7%)	84 (2.1%)	87 (2.0%)	89 (1.6%)
C-reactive protein >3	6721 (37.7%)	1838 (46.3%)	1792 (41.6%)	1959 (35.9%)

Abbreviations:

[*] ACR: Albumin to Creatinine Ratio

[†] DBP: diastolic blood pressure

[ǂ] eGFR: estimated glomerular filtration rate

[§] HDL: high-density lipoprotein

[?] SBP: systolic blood pressure

[#] SD: Standard deviation

Table 2.

Hazard Ratios for incident HF event according to DASH diet score

A. All				
Model	Q1 (Score 8–20)	Q2 (Score 21–23)	Q3 (Score 24–27)	Q4 (Score 28–40)
N (events)	4,203 (200)	4,559 (179)	5,764 (224)	4,330 (164)
Event Rate, person-years	53/10,000	43/10,000	41/10,000	40/10,000
1*	1.00 (ref)	0.68 (0.55,0.83)	0.62 (0.51,0.75)	0.55 (0.45,0.68)
2†	1.00 (ref)	0.73 (0.59,0.89)	0.75 (0.61,0.91)	0.70 (0.57,0.88)
3‡	1.00 (ref)	0.69 (0.56,0.85)	0.71 (0.58,0.87)	0.73 (0.58,0.92)
B. Age<65				
Model	Q1 (Score 8–20)	Q2 (Score 21–23)	Q3 (Score 24–27)	Q4 (Score 28–40)
N (events)	2,624 (82)	2,427 (45)	2,865 (44)	1,927 (22)
Event Rate, person-years	35/10,000	20/10,000	16/10,000	12/10,000
1*	1.00 (ref)	0.54 (0.38,0.78)	0.43 (0.30,0.62)	0.31 (0.19,0.49)
2†	1.00 (ref)	0.60 (0.41,0.86)	0.53 (0.36,0.77)	0.42 (0.26,0.67)
3‡	1.00 (ref)	0.56 (0.39,0.81)	0.54 (0.37,0.79)	0.48 (0.30,0.78)
C. Age 65–74				
Model	Q1 (Score 8–20)	Q2 (Score 21–23)	Q3 (Score 24–27)	Q4 (Score 28–40)
N (events)	1,145 (7)	1,457 (80)	1,941 (77)	1,579 (62)
Event Rate, person-years	77/10,000	60/10,000	41/10,000	40/10,000
1*	1.00 (ref)	0.78 (0.57,1.07)	0.54 (0.39,0.74)	0.51 (0.37,0.72)
2†	1.00 (ref)	0.87 (0.63,1.18)	0.65 (0.48,0.90)	0.67 (0.48,0.94)
3‡	1.00 (ref)	0.79 (0.58,1.08)	0.57 (0.42,0.79)	0.65 (0.46,0.92)
D. Age≥ 75				
Model	Q1 (Score 8–20)	Q2 (Score 21–23)	Q3 (Score 24–27)	Q4 (Score 28–40)
N (events)	434 (39)	675 (54)	958 (103)	824 (80)
Event Rate, person-years	113/10,000	98/10,000	126/10,000	113/10,000
1*	1.00 (ref)	0.83 (0.55,1.25)	1.07 (0.74,1.55)	0.96 (0.66,1.42)
2†	1.00 (ref)	0.81 (0.54,1.23)	1.19 (0.82,1.72)	1.13 (0.77,1.66)
3‡	1.00 (ref)	0.81 (0.54,1.24)	1.21 (0.83,1.76)	1.12 (0.75,1.66)

*Model 1 adjusts for total energy intake, age, sex, and race

†Model 2 adjusts for Model 1 plus other socio-demographic factors such as geographic region, annual family income (< vs. ≥ $20,000/year), educational achievement (< vs. ≥ high school diploma), health insurance, marital status, cigarette smoking, alcohol consumption, physical activity

‡Model 3 adjusts for Model 2 plus potentially-mediating physiologic parameters such as systolic blood pressure, diastolic blood pressure, body mass index, high-density lipoprotein cholesterol, total cholesterol, triglycerides, estimated glomerular filtration rate, natural log-transformed C-reactive protein, urine albumin-to-creatinine ratio, diabetes, history of coronary heart disease, history of atrial fibrillation, and left ventricular hypertrophy