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Dietary Patterns and Indicators of Cognitive Function

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Abstract

Importance: Healthier diets are generally believed to benefit cognitive health; however, the evidence remains inconsistent, and a systematic examination of multiple healthy dietary patterns within a same context is lacking.

Objectives: To evaluate the associations of six healthy patterns with subjective cognitive decline (SCD) and objective cognitive function.

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Contributors: KB and CY conceived and designed the study. HC conducted statistical analysis and wrote the first draft of the paper. MC, MHF, AT, DD, and AA provided statistical expertise. WCW, DD, and CY obtained funding. All authors contributed to the interpretation of the results and critical revision of the manuscript for important intellectual content and approved the final version of the manuscript. KB and CY are the guarantors. The corresponding author attests that all listed authors meet authorship criteria and that no others meeting the criteria have been omitted.

Ethical approval: The Nurses' Health Study, Nurses' Health Study II and the Health Professionals Follow-up Study were approved by the institutional review boards at Brigham and Women's Hospital and Harvard T H Chan School of Public Health. The return of the completed self-administered questionnaire was considered to imply informed consent.

Design and Setting: Prospective cohort study based on the Nurses' Health Study (NHS, 1986–2014), NHSII (1991–2017), and the Health Professionals Follow-up Study (HPFS, 1986–2012). Data analyses were performed from September 2024 to November 2025.

Participants: 159,347 women and men with available data on diet and cognitive function.

Exposures: Six dietary pattern scores, including the Alternate Healthy Eating Index (AHEI)-2010, the Dietary Approaches to Stop Hypertension (DASH) diet score, the healthful plant-based diet index (hPDI), the planetary health diet index (PHDI), and the reversed empirical dietary indices for hyperinsulinemia (rEDIH) and inflammatory pattern (rEDIP).

Main outcomes and measures: SCD was assessed using seven questions on perceived cognitive changes. Cognitive function was objectively measured via telephone in the NHS.

Results: Among the study participants (mean age: 44.3 years, standard deviation: 9.3 years; 82.6% women), higher adherence to all six dietary patterns was associated with a lower SCD risk. DASH diet showed the strongest magnitude (risk ratio [RR] comparing 90th vs 10th percentile of adherence: 0.59, 95% CI: 0.57–0.62), followed by the hPDI (RR: 0.76, 0.65–0.85), rEDIH (RR: 0.76, 0.73–0.80), PHDI (RR: 0.80, 0.75–0.86), AHEI-2010 (RR: 0.84, 0.80–0.89), and rEDIP (RR: 0.89, 0.85–0.93). Higher DASH diet score at ages 45–54 years showed the strongest association with SCD. Higher adherence to the DASH diet also showed the strongest association with a higher objectively measured global cognition (mean z-score difference comparing 90th vs 10th percentile: 0.053, 95% CI: 0.015–0.091). Key food groups associated with better cognitive function included higher vegetable and fish intake and lower red and processed meats intake.

Conclusions and Relevance: Healthy diets, exemplified by the DASH diet for blood pressure control and diets with lower hyperinsulinemia and inflammation potentials, were associated with a lower SCD risk and better cognitive function. These findings underscore the importance of a healthy diet for maintaining long-term cognitive health.

INTRODUCTION

Dementia impacts the well-being of older adults,[1,2] and the number of individuals living with dementia is projected to reach 150 million by 2050.[3] Early prevention through healthy lifestyle modifications is crucial,[4] among which healthy diet has received increasing attention.[5,6] For example, previous studies in the Nurses' Health Study (NHS) and the Health Professionals Follow-up Study (HPFS) reported that higher intake of red and processed meat was associated with worse cognitive outcomes,[7] and a healthy diet was inversely related to future cognitive impairment.[8] However, the role of diet on cognitive health is still uncertain,[9,10] and further investigations are needed to understand the roles of healthy dietary patterns in early stages of cognitive aging.

Discrepancies in existing studies may arise from differences in study design, study populations, follow-up durations, and exposure and outcome measurements. For instance, the UK Whitehall II study found no significant association between the Alternate Healthy Eating Index (AHEI) and dementia risk,[11] whereas a meta-analysis reported protective associations of a healthy diet.[10] Additionally, short follow-up periods and the lack of repeated dietary assessments to capture long-term intake limit causal inference, as the extended preclinical phase of dementia may lead to behavioral changes and memory impairments that confound dietary reporting.[5,12] Furthermore, limited evidence exists regarding the cognitive effects of some emerging dietary patterns, such as plant-based and planetary health diets.[13–15] Lastly, few studies have systematically evaluated multiple healthy dietary patterns within the same population, which is essential for optimizing dietary strategies for promoting healthy cognitive aging.[16]

To address these research gaps, we systematically explored the associations of adherence to six established healthy dietary patterns with subjective cognitive decline (SCD), an early indicator of cognitive problems preceding detectable deficits,[8,17,18] as well as objectively measured cognitive function.

METHODS

Study population

This study was based on three ongoing cohorts: The NHS enrolled 121,700 female nurses aged 30–55 years in 1976.[19] The HPFS enrolled 51,529 male health professionals aged 40–75 years in 1986. The NHSII enrolled 116,429 female nurses aged 25–42 years in 1989. All three cohorts mailed biennial questionnaires to participants, collecting data on socioeconomic status, lifestyle, medical history, and other health-related factors.

The study baseline was defined as 1986 for NHS and HPFS and 1991 for NHSII, when detailed semi-quantitative food frequency questionnaire (FFQ) data was available for calculation of the diet scores. We included participants who agreed to share their data and excluded participants with extreme energy intake (<600 or >3500 kcal/d for women and <800 or >4200 kcal/d for men), missing data on dietary pattern scores or SCD, or a diagnosis of Parkinson's disease before SCD assessment (eFigure 1 in the Supplement). All three cohorts were approved by the Brigham and Women's Hospital and Harvard T.H. Chan School of Public Health. The return of the completed self-administered questionnaire was considered to imply informed consent. This study followed the STROBE guidelines.

Dietary patterns

Diet was assessed using FFQs every four years (NHS: 1986–2006; NHSII: 1991–2011; HPFS: 1986–2002), which capture habitual long-term dietary intake and have been extensively validated.[20–22] Six dietary patterns were included (eTable 1 in the Supplement): the AHEI-2010, the Dietary Approaches to Stop Hypertension (DASH) diet score, the healthful plant-based diet index (hPDI), the planetary health diet index (PHDI), and the reversed empirical dietary indices for hyperinsulinemia (rEDIH) and inflammatory pattern (rEDIP).[23] Details of the scoring criteria are described in eMethods 3 in the Supplement. These scores showed a wide range of correlations (Spearman's ρ: 0.25–0.82, eFigure 2 in the Supplement), reflecting related yet different measured dietary patterns. To reduce random measurement error, we calculated cumulative average dietary scores from repeated FFQ assessments over follow-up, as a more stable representation of long-term dietary intake (eTable 2 in the Supplement). Because diet could change due to early cognitive symptoms, we stopped updating the scores 6 years before SCD assessments and 5 years before objective cognitive assessment.[8,24]

Subjective cognitive decline

The primary outcome is SCD, assessed in 2012 and 2014 in the NHS, 2017 in the NHSII, and 2008 and 2012 in the HPFS using 6 (HPFS) or 7 (NHS and NHSII) yes/no questions on self-perceived recent changes in memory, executive function, attention, and visuospatial skills.[25] As an early signal before measurable impairment, SCD tends to be more informative and sensitive in highly educated people.[26] The SCD score showed strong and linear associations with objective cognitive test scores.[25] To represent late-life SCD status, we averaged scores for participants with repeated measurements. Domain-specific SCD was defined as any difficulty in each domain.

Objective cognitive function

In 1995, stroke-free NHS participants aged ≥70 years were invited to telephone-based cognitive assessments, followed by three additional rounds of biennial follow-up assessments (round-to-round retention rates >90%).[27] While baseline characteristics were generally similar, respondents were less diverse and appeared more health conscious compared with eligible non-respondents, with higher multivitamin/aspirin use and slightly higher energy intake (eTable 3 in the Supplement). Trained nurses assessed global cognitive function using the Telephone Interview of Cognitive Status (TICS),[28] supplemented by immediate and delayed recalls of the East Boston Memory Test (for verbal memory), animal fluency test (for verbal fluency), and digit span backward (for working memory) in the follow-up assessments.[29] This cognitive battery showed a high correlation with a face-to-face version ($r = 0.81$) and high inter-interviewer reliability ($r > 0.95$).[29] Scores were averaged across the rounds of tests.[30]

Statistical analysis

Participant characteristics are described using the age-standardized means (standard deviation, SD) for continuous variables and percentages for categorical variables. We performed analyses within each cohort separately and pooled the estimates using random-effects meta-analysis.

We assessed the associations of the diet scores and food groups with SCD status using Poisson regression, treating the SCD score as a count variable. Risk ratios (RRs) and 95% confidence intervals (CIs) are presented for 3-unit increments in SCD, as ≥3 positive SCD questions define severe SCD.[31] Continuous diet scores were scaled by the 10th-90th percentile range (the difference between the 90th and 10th percentile values), so the estimates represented comparisons between individuals at the 90th percentile and those at the 10th percentile.[16] We also modeled them as categorical variables (quintiles) to assess and compare potential non-linear associations. Model 1 was adjusted for age at baseline (1986 for NHS and HPFS and 1991 for NHSII) and total energy intake. Model 2 was adjusted for all other covariates (eMethods 2 in the Supplement). We used logistic regression to assess the associations with domain-specific SCD (as binary outcome variables). For the objective cognitive tests, we assessed the associations of the average diet scores in 1986 and 1990 with global and domain-specific cognitive test scores in 1995–2001 using linear regression. We assessed the associations of food groups with global and domain-specific SCD and objective cognitive test scores using the same approach, standardizing the food group intake levels as the 90th vs. 10th percentile variables.

We conducted several pre-planned exploratory analyses, including latency analyses using dietary scores assessed 6–26 years before SCD measurement, across life stages (5-year age groups), and subgroup analyses by demographic, lifestyle, and genetic factors.[16,23] We also performed pre-specified sensitivity analyses, as described in the eMethods 3 in the Supplement.

We performed the analyses using SAS 9.4 and R 4.3.0 from September 2024 to November 2025. All tests are two-tailed. The α-level was set at 0.05. Despite the exploratory nature of the present study, we addressed potential multiplicity using false-discovery-rate correction for each hypothesis family (eMethods 3 in the Supplement).

RESULTS

Population characteristics

This study included 62,412 women from the NHS, 69,148 women from the NHSII, and 27,787 men from the HPFS (Table 1). Participants with higher diet scores, indicating healthier diets, had lower BMI, higher physical activity levels, and a lower prevalence of health conditions.

Dietary patterns and subjective cognitive decline

Higher scores across six dietary patterns were associated with a lower risk of global SCD (Table 2 and Figure 1). For instance, the RRs for the increasing quintiles of the DASH score were 1.00 (reference), 0.91 (95% CI: 0.85, 0.97), 0.78 (95% CI: 0.73, 0.83), 0.74 (95% CI: 0.69, 0.79), and 0.59 (95% CI: 0.55, 0.63), respectively, and the RR for increment from the 10th to the 90th percentiles was 0.59 (95% CI: 0.57, 0.62). Other dietary patterns, i.e., hPDI (RR: 0.76, 95%CI: 0.68, 0.85), rEDIH (RR: 0.76, 95%CI: 0.73, 0.80) PHDI (RR: 0.80, 95%CI: 0.75, 0.86), AHEI-2010 (RR: 0.84, 95%CI: 0.80, 0.89), and rEDIP (RR: 0.89, 95%CI: 0.85, 0.93), also showed significant associations. The comparisons of these estimates are presented in eFigure 2 in the Supplement. We did not detect substantial departures from linearity for any diet score (eFigure 3 in the Supplement). The associations of combinations of higher diet scores (above the median) with SCD in later life suggest additive benefits of adhering to multiple healthy dietary patterns for cognitive health (Figure 1B). Higher adherence to each dietary patterns was associated with lower risk of memory SCD (eTable 4 in the Supplement).

Dietary patterns and objective cognitive function

In the NHS, we assessed the associations of the dietary patterns with the global and domain-specific cognitive test scores (Figure 2 and eTable 5 in the Supplement). Higher diet scores for each of the dietary patterns, except for hPDI and PHDI, were associated with higher cognitive z-scores. For instance, compared with those at the 10th percentile of the DASH score, participants at the 90th percentile, averagely. had a 0.053 (95% CI: 0.015, 0.091) higher global cognition z-score (equivalent to 0.76 years younger in cognitive aging), a 0.044 (95% CI: 0.010, 0.079) higher verbal fluency z-score (0.87 years younger), and a 0.049 (95% CI: 0.015, 0.082) higher working memory z-score (1.37 years younger).

Food group intake and subjective cognitive decline and objective cognitive function

The associations of the food group intake levels with cognitive outcomes are shown in Figure 3 and eTable 6 in the Supplement. Green-leafy, yellow, and other vegetables, which were positively correlated with most dietary scores, were significantly associated with better cognition. Conversely, fried, but not non-fried, potatoes were associated with a higher risk of SCD and worse objective cognitive performance. Fruits were associated with a lower risk of SCD in the three cohorts but lower verbal memory z-score in the NHS. Unexpectedly, a higher intake of nuts and seeds was associated with higher SCD risk. Red and processed meats and eggs were associated with worse cognitive outcomes, whereas fish intake was related to better cognitive function. Wine, tea, and salad dressings were associated with better cognition, while sweetened beverages and sweets were associated with worse cognition.

Latency and life stage-specific associations of diet scores with SCD

When assessing the diet scores 6 to 26 years before SCD assessment (eFigure 4 and eTable 7 in the Supplement), higher DASH diet scores and hPDI were consistently associated with a lower risk of SCD at all time points, while for the remaining dietary patterns, the risk estimates were attenuated with longer intervals between dietary and SCD assessments. The association between average dietary pattern scores at different life stages and SCD risk assessed in later life is shown in eFigure 5 in the Supplement. In the HPFS and NHS, DASH scores at 45–54 years of age tended to have the strongest associations with later-life SCD.

Subgroup and sensitivity analyses

We observed similar associations in the subgroups defined by cohort, age, BMI, and income (eFigure 6 and eTable 8 in the Supplement). While the magnitude of association differed moderately by certain lifestyle factors (e.g., stronger association for the DASH diet in the low-alcohol-intake group), the interaction tests did not reach statistical significance. The associations between higher diet scores with lower SCD risk persisted in sensitivity analyses with alternative outcome definition, exclusion criteria, exposure update window, and accounting for potential selection bias due to differential survival (eTable 9 in the Supplement).

DISCUSSION

In three US-based cohorts, adherence to healthy dietary patterns, exemplified by the DASH diet, was generally associated with a lower risk of SCD and better objective cognitive function in a dose-response manner. Higher intake of vegetables, fish, and moderate wine consumption contributed to the observed associations, whereas red and processed meats, fried potatoes, and sugary beverages were associated with poorer cognition. These findings suggest that a healthy diet may benefit cognitive health in future life.

These findings accord with prior work on cognitive benefits of healthy dietary patterns.[32] In a community-based study, higher HEI-2015 was associated with a lower risk of dementia.[33] In the NHS, higher DASH and AHEI-2010 scores were previously associated with a lower risk of SCD,[8] which was confirmed and expanded on in our study. Our findings generally support the role of a healthy diet, manifested by six dietary patterns, in benefiting cognitive health. For example, participants at the 90th versus 10th percentile of the DASH score (≈10.9-point difference, corresponding to improving adherence in 2~3 dietary components) had 41% lower odds of SCD. While the subgroup analysis suggested potential variation in strength of the association by certain lifestyle factors, such as physical activity, the study was not designed or powered to detect significant interaction for individual potential modifiers. Further studies with larger sample sizes are needed to reveal modifiers of the diet-cognition association,[34] and large-scale long-term clinical trials are needed to fully reveal the cognitive effects of the healthy diet.[35–37]

While the healthy dietary patterns were generally associated with better cognitive health, they have distinct attributes. The blood pressure-targeted DASH diet, which showed strong associations in our study, is featured by higher intake of vegetables,[38] nuts,[39] and whole grains,[40] while limiting sugar and sodium. While direct evidence for the mediation role of hypertension in diet-cognition pathways remains limited, our findings aligned with prior literature on the cognitive benefits of blood pressure control and cognitive health.[41,42] The hPDI and PHDI were designed to promote both environmental sustainability and human health,[13,15] but evidence on their cognitive effects remains limited.[43] Our study revealed that higher hPDI and PHDI were associated with better cognition. Furthermore, our study explored data-driven dietary patterns through rEDIH and rEDIP, which capture dietary patterns that predict hyperinsulinemia and chronic inflammation. While their associations might be diluted by components that are less cognition-specific, our findings generally supported the role of insulin and inflammatory pathways in cognitive health.

Although association magnitudes for dietary pattern varied, these differences should be interpreted cautiously. The scores are moderately to highly correlated, and observed differences may reflect underlying population characteristics rather than biological distinctions. Additionally, the potential for inflated Type I error due to multiple testing of the secondary analyses in the observational study warrants cautious interpretation and desirability of future intervention studies to confirm these findings. Based on our examinations, further studies may devise and optimize diets for cognitive health and design feasible intervention approaches.

The temporal and life stage-specific associations observed in our study inform the potential timing of dietary effects on brain health.[44] The DASH diet was consistently associated with SCD risk even when measured up to 26 years before the SCD assessments and had robust protective associations at various ages, particularly in midlife (45–54 years). This pattern minimizes the possibility of reverse causation, although it could also reflect period or survivor effects. This aligns with emerging evidence that midlife is a key period for modifiable risk factors influencing late-life cognitive outcomes, including diabetes and hypertension.[1] Further studies are needed to determine the optimal timing for dietary interventions and whether improvements in later life can still yield significant benefits for brain health.

The strengths of this study included the large sample size, the long-term follow-up and the repeated dietary measurements that enabled us to assess the temporal and life stage-specific associations, and a wide range of dietary patterns investigated. We aligned dietary assessments and cognitive evaluations to the same calendar periods within each cohort, thereby minimizing the potential for secular trends to confound within-cohort associations. Nevertheless, several limitations should be noted. First, we focused on SCD and objectively measured objective cognitive function, which represents the early-stage symptoms of neurodegeneration. As SCD reflects perceived rather than objectively measured cognitive change, it must be self-reported and may be influenced by differences in health awareness or reporting tendencies, despite prior validation of this measure.[25] The weaker associations with objective cognitive tests may reflect range restriction from limiting testing to older individuals (≥70 years) with more homogeneous dietary patterns and cognitive function, and potential ceiling effects in this highly educated subsample. SCD and objective tests capture complementary aspects of cognitive health, and associations with clinical endpoints such as AD diagnosis should be further investigated. Second, while we have carefully adjusted the models for potential confounding factors and used long-term latency analyses to reduce reverse causation, residual confounding cannot be ruled out. The attenuation in associations observed from minimally to fully adjusted models, without further change when accounting for other sources of bias, likely reflects confounding by healthy lifestyles and socioeconomic status. Individuals with healthier lifestyles may have both healthier diet and diet-independent dementia risk, although healthy diet could also be a mediator. Third, measurement error and self-report bias, such as social desirability bias, in dietary assessment are inevitable,[45] and dietary behaviors have changed over time. To address these, we used long-term repeated FFQs before outcome assessment and adjusted for a wide range of lifestyle and socioeconomic factors that may correlate with healthy reporting tendencies. Finally, the cohorts consist predominantly of White, highly educated health professionals, and long-term societal and environmental changes may influence the generalizability of the findings. However, this relative homogeneity may increase internal validity by reducing confounding, because they should have good baseline cognitive ability, and prior work in these cohorts on diet and other health outcomes has yielded effect estimates similar to those in more diverse populations.[16,46]

In conclusion, healthy diets, including a priori defined and mechanism-informed dietary patterns, were generally associated with a lower risk of SCD and better objectively measured cognitive function. Better adherence to a healthy diet, exemplified by the DASH diet, from mid-adulthood, exhibited stronger associations. These findings support the importance of healthy eating as part of midlife brain-health strategies and motivate pragmatic and implementation research to translate these findings into scalable programs.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data sharing:

Because of participant confidentiality and privacy concerns, requests to access NHS/NHSII/HPFS data must be submitted in writing. According to standard controlled access procedures, applications to use NHS/NHSII/HPFS resources will be reviewed by the External Collaborations Committee to verify that the proposed use maintains the protection of the privacy of participants and the confidentiality of the data. Investigators wishing to use NHS/NHSII/HPFS data are asked to submit a brief description of the proposed project (go to https://www.nurseshealthstudy.org/researchers (contact email: nhsaccess@channing.harvard.edu) and https://sites.sph.harvard.edu/hpfs/for-collaborators/ for details.

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KEY POINTS

Question:

Are healthy dietary patterns associated with lower risk of subjective cognitive decline and better objectively measured cognitive function?

Findings:

In a systematic evaluation of six dietary patterns among ~160,000 participants, greater adherence to a healthy diet, exemplified by Dietary Approaches to Stop Hypertension (DASH) diet, was associated with lower risk of subjective cognitive decline and better objectively measured cognitive function. The associations were most pronounced when the diet was followed during mid-adulthood (ages 45–54).

Meaning:

A healthy diet, such as the DASH diet, was associated with early indicators of cognitive aging, which underscore the importance of a healthy diet for maintaining long-term cognitive health.

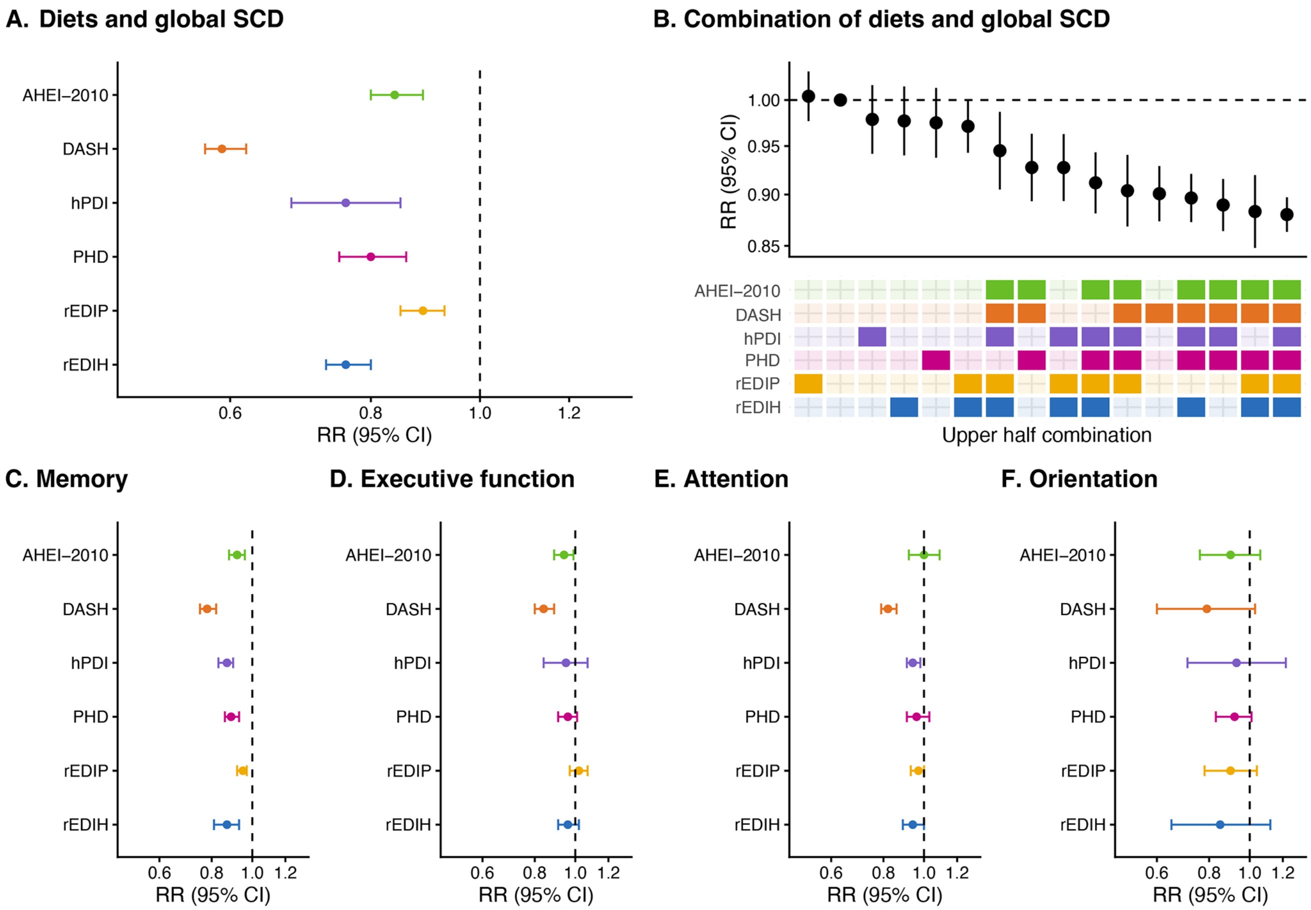

Figure 1.

Associations of dietary pattern scores with global and domain-specific subjective cognitive decline

In panels A and C-F, points indicate risk ratio (RRs) for each 3-unit increment in the subjective cognitive decline (SCD) score or for binomial impairment variables for cognitive domains comparing the 90th to the 10th percentile of dietary scores, and error bars indicate 95% confidence intervals (CIs). RRs below 1 indicate lower risk of subjective cognitive decline. In panel B, the points show RRs of SCD on the y axis, bars reflecting 95% CIs, and adherence to the six different dietary patterns below the x-axis. The shading for each dietary pattern reflects the strength of adherence, with adherence for dietary indices in the upper half in block colors and those in the lower half in the light shaded colors. This combination-based analysis illustrates how multiple healthy dietary patterns cumulatively relate to SCD.

The models were adjusted for age at baseline (1986 for NHS and HPFS and 1991 for NHSII), total energy intake, race, highest education level (for NHS and NHSII), highest education level of spouse (NHS), profession (HPFS), census median household income, smoking pack-years, alcohol intake (gram/day), body mass index, high blood pressure, elevated cholesterol, diabetes, stroke, myocardial infarction, angina, coronary artery bypass surgery, cancer, depression, family history of dementia, aspirin use, multivitamin use, and post-menopausal hormone use (in NHS and NHSII).

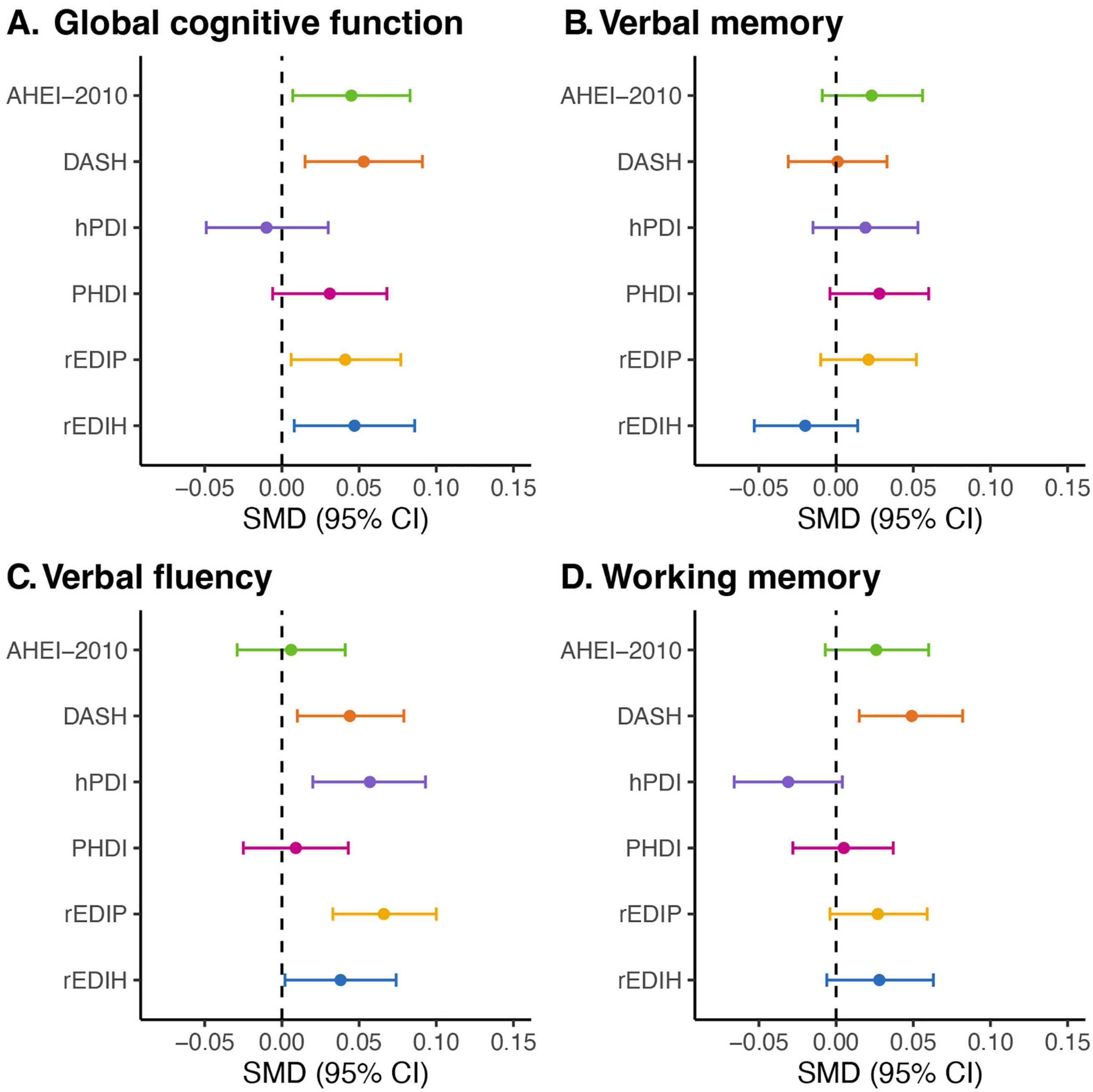

Figure 2.

Associations of average dietary pattern scores with global and domain-specific objective cognitive function z-scores in the Nurses' Health Study

SMD: standardized mean difference; CI: confidence interval

Points indicate beta estimates for z-scores in objective cognitive function tests comparing the 90th to the 10th percentile of dietary scores. Error bars reflect the 95% confidence intervals. Larger estimates indicate better cognitive function. The model was adjusted for age at baseline (1986), total energy intake, race, highest education level, highest education level of spouse, census median household income, smoking pack-years, alcohol intake (gram/day), body mass index, high blood pressure, elevated cholesterol, diabetes, stroke, myocardial infarction, angina, coronary artery bypass surgery, cancer, depression, family history of dementia, aspirin use, multivitamin use, and post-menopausal hormone use.

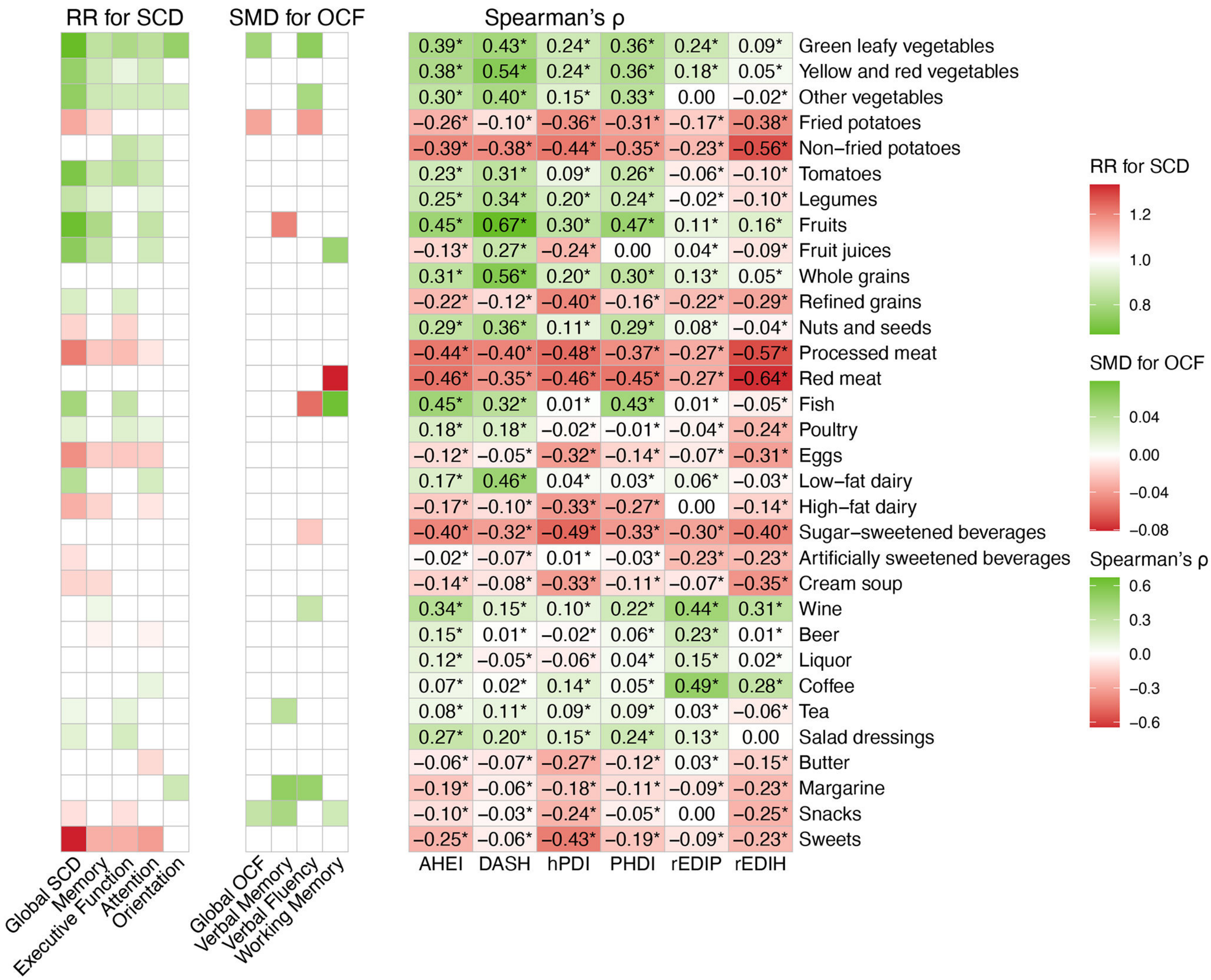

Figure 3.

Associations of average food group intake with cognitive outcomes and their correlations with dietary pattern scores

SCD: subjective cognitive decline; OCF: objective cognitive function; VM: verbal memory; VF: verbal fluency; WM: working memory; SMD: standardized mean difference.

For the associations of average food group intake (the 90th vs. the 10th percentile) with cognitive outcomes (left two heatmaps) with false-discovery-rate (FDR)-corrected *P*-values <0.05, we used green color to indicate lower SCD score or higher OCF scores and red color for unfavorable outcomes. White color represents associations with FDR-corrected *P*-values ≥0.05. The Spearman's ρs for correlations between food groups and dietary pattern scores (the right heatmap) were calculated from the combined dataset of three cohorts. Green color indicates positive correlations, and red color indicates negative correlations, and correlations with FDR-corrected *P*-values <0.05 were marked with an asterisk in the box.

Table 1

Population characteristics by diet scores in the pooled dataset of the Nurses' Health Study (NHS), NHSII, and Health Professional Follow-up Study (HPFS)

Variable	AHEI		DASH		hPDI	
	Q1 (n=31,868)	**Q5 (n=31,868)**	**Q1 (n=31,929)**	**Q5 (n=32,028)**	**Q1 (n=31,846)**	**Q5 (n=31,994)**
Age at baseline, years, mean (SD)	43.4 (9.3)	45.2 (9.1)	43.1 (8.6)	45.7 (9.8)	43.1 (9.1)	45.6 (9.3)
Male, %	18.5	16.4	18.8	15.9	18.9	16.1
Race						
- White, %	96.9	95.5	95.7	96.4	96.9	95.3
- Black, %	1.1	1.4	1.5	1.2	1.0	1.5
- Other, %	2.0	3.2	2.8	2.5	2.1	3.2
Married, %	71.3	73.4	71.1	73.2	73.1	71.5
Total energy intake, mean (SD)	1903.8 (483.0)	1771.6 (448.0)	1701.5 (478.9)	1975.2 (448.5)	2095.2 (466.7)	1597.3 (407.8)
Smoking, pack-years, mean (SD)	9.3 (16.4)	6.7 (11.6)	11.1 (17.4)	5.6 (10.8)	8.1 (15)	7.5 (12.8)
Alcohol intake, gram/day, mean (SD)	4.9 (10.6)	7.3 (7.5)	6.3 (10.4)	5.9 (7.9)	5.7 (9.1)	6.2 (8.7)
BMI, mean (SD)	26.6 (5.9)	24.4 (4.5)	26.4 (5.8)	24.6 (4.8)	26.4 (5.9)	24.7 (4.7)
Physical activity, MET-hours/week, mean (SD)	15.6 (15.6)	30.7 (23.9)	15.3 (15.4)	31.1 (24.2)	17.9 (17.1)	28.5 (23.9)
Census tract median household income, $, mean (SD)	58703.1 (19323.9)	71052.8 (25968.8)	60397.1 (20725.4)	68429.3 (24858.8)	62193.6 (21283.1)	67650.2 (24787.6)
High Blood Pressure, %	51.5	40.1	50.8	40.7	50.4	41.7
Elevated Cholesterol, %	61.7	57.1	62.6	56.4	60.8	58.4
Diabetes, %	9.3	5.2	8.9	5.5	8.5	6.1
Cardiovascular disease, %	6.1	4.7	5.9	4.8	5.7	5.0
Stroke, %	1.3	0.9	1.3	0.9	1.2	1.0
Myocardial infarction, %	1.9	1.5	1.9	1.5	1.7	1.5
CABG, %	4.7	4.4	4.8	4.4	4.2	4.5
Angina, %	3.0	2.5	2.6	2.5	2.7	2.5
Cancer, %	11.7	12.4	12.0	12.4	12.2	11.9
Depression, %	19.4	16.8	19.0	16.9	18.4	17.0
Multivitamin use, %	58.9	68.4	55.3	71.0	60.8	67.5
Aspirin use, %	40.5	37.6	40.3	37.6	41.8	37.1

Variable	PHDI		rEDIP		rEDIH	
	Q1 (n=31,868)	Q5 (n=31,868)	Q1 (n=31,868)	Q5 (n=31,868)	Q1 (n=31,868)	Q5 (n=31,868)
Age at baseline, years, mean (SD)	43.2 (9.2)	45.4 (9.2)	43.8 (9.2)	44.3 (8.8)	42.9 (8.6)	45.5 (9.5)
Male, %	18.7	16.2	19.2	16.3	18.0	17.6
Race						
- White, %	97.2	94.4	93.9	97.9	96	96.6
- Black, %	0.9	1.7	2.2	0.4	1.5	0.8
- Other, %	1.9	3.9	4.0	1.8	2.6	2.6
Married, %	68.0	74.5	71.2	73.6	72.2	71.6
Total energy intake, mean (SD)	1913.5 (521.7)	1754.6 (427.6)	1941.7 (505.7)	1829.7 (462.5)	2179 (476.2)	1600.4 (416.5)
Smoking, pack-years, mean (SD)	9.4 (16.3)	6.6 (11.8)	7.7 (14.5)	9.4 (14.6)	8.5 (15.3)	8.2 (13.5)
Alcohol intake, gram/day, mean (SD)	5.1 (9.1)	7.0 (8.9)	3.0 (6.3)	12.8 (12.6)	4.7 (8.5)	10.1 (11.3)
BMI, mean (SD)	26.3 (5.9)	24.5 (4.6)	27.5 (6.3)	24.2 (4.2)	27.6 (6.3)	23.8 (4.0)
Physical activity, MET-hours/week, mean (SD)	16.4 (16.7)	29.9 (23.9)	19 (18.8)	26.2 (22.3)	18.9 (18.4)	27.5 (23.4)
Census tract median household income, $, mean (SD)	60434.2 (20300.5)	69423.6 (25496)	60338.1 (20827.3)	68790.8 (24980.6)	59499.9 (20131.1)	70426.8 (25747.5)
High Blood Pressure, %	50	41.4	56.1	40.2	54.7	38.5
Elevated Cholesterol, %	60.5	58.6	65.4	55.4	63.8	55.4
Diabetes, %	8.6	6.0	13.9	3.1	13.4	3.1
Cardiovascular disease, %	6.0	5.0	7.3	3.9	7.1	4.2
Stroke, %	1.3	0.9	1.3	0.8	1.4	0.9
Myocardial infarction, %	1.8	1.6	2.4	1.2	2.1	1.4
CABG, %	4.4	4.6	5.9	3.6	5.2	4.0
Angina, %	2.9	2.6	3.7	2.0	3.7	2.1
Cancer, %	11.8	12.3	12.2	12.5	11.8	12.3
Depression, %	19.2	16.6	20.6	16.8	20.8	16.5
Multivitamin use, %	59.3	68.5	60.7	67.2	61.1	66.0
Aspirin use, %	39.5	37.9	40.9	39.7	42.5	37.2

The distributions of all variables other than age were age standardized. The diet scores were categorized into quintiles by cohort. All dietary scores and covariates, except for age, were measured as the averages across study waves for continuous variables and as the prevalence at the latest dietary assessment for categorical variables.

Table 2

Associations of average dietary pattern scores (in quintiles and comparing the 90th vs. the 10th percentile) with global subjective cognitive decline

Diet	Model	Risk ratio (95% CI)						*P*-trend	FDR-corrected *P*-trend
		Q1	Q2	Q3	Q4	Q5	90^{th} v. 10^{th} percentile		
AHEI-2010	Age- and energy-adjusted	1.00 (Ref)	0.98 (0.88, 1.09)	**0.88 (0.83, 0.94)**	**0.76 (0.71, 0.81)**	**0.69 (0.65, 0.73)**	**0.66 (0.60, 0.72)**	<.001	<.001
	MV-adjusted	1.00 (Ref)	1.03 (0.90, 1.18)	0.98 (0.87, 1.11)	0.88 (0.77, 1.01)	0.89 (0.73, 1.09)	**0.84 (0.80, 0.89)**	<.001	<.001
DASH	Age- and energy-adjusted	1.00 (Ref)	**0.85 (0.76, 0.96)**	**0.69 (0.63, 0.77)**	**0.63 (0.53, 0.74)**	**0.46 (0.36, 0.59)**	**0.47 (0.38, 0.57)**	<.001	<.001
	MV-adjusted	1.00 (Ref)	**0.91 (0.85, 0.97)**	**0.78 (0.73, 0.83)**	**0.74 (0.69, 0.79)**	**0.59 (0.55, 0.63)**	**0.59 (0.57, 0.62)**	<.001	<.001
hPDI	Age- and energy-adjusted	1.00 (Ref)	0.98 (0.92, 1.04)	**0.89 (0.82, 0.97)**	**0.80 (0.74, 0.85)**	**0.65 (0.56, 0.76)**	**0.66 (0.55, 0.79)**	<.001	<.001
	MV-adjusted	1.00 (Ref)	1.00 (0.94, 1.07)	0.93 (0.85, 1.00)	**0.87 (0.81, 0.93)**	**0.77 (0.70, 0.86)**	**0.76 (0.68, 0.85)**	<.001	<.001
PHDI	Age- and energy-adjusted	1.00 (Ref)	**0.90 (0.84, 0.97)**	**0.81 (0.69, 0.95)**	**0.78 (0.71, 0.86)**	**0.65 (0.60, 0.69)**	**0.66 (0.60, 0.73)**	<.001	<.001
	MV-adjusted	1.00 (Ref)	0.96 (0.84, 1.09)	0.90 (0.73, 1.12)	**0.90 (0.84, 0.96)**	**0.81 (0.66, 0.99)**	**0.80 (0.75, 0.86)**	<.001	<.001
rEDIP	Age- and energy-adjusted	1.00 (Ref)	**0.88 (0.78, 0.99)**	**0.81 (0.71, 0.92)**	**0.78 (0.72, 0.84)**	**0.72 (0.65, 0.78)**	**0.77 (0.71, 0.84)**	<.001	<.001
	MV-adjusted	1.00 (Ref)	**0.93 (0.87, 0.99)**	**0.89 (0.83, 0.95)**	**0.90 (0.84, 0.96)**	**0.82 (0.76, 0.88)**	**0.89 (0.85, 0.93)**	<.001	<.001
rEDIH	Age- and energy-adjusted	1.00 (Ref)	**0.85 (0.73, 0.98)**	**0.74 (0.67, 0.82)**	**0.69 (0.64, 0.75)**	**0.59 (0.55, 0.63)**	**0.63 (0.58, 0.68)**	<.001	<.001
	MV-adjusted	1.00 (Ref)	0.92 (0.81, 1.04)	**0.85 (0.76, 0.94)**	**0.83 (0.76, 0.90)**	**0.74 (0.68, 0.81)**	**0.76 (0.73, 0.80)**	<.001	<.001

The age- and energy-adjusted model was adjusted for age at baseline (1986 for NHS and HPFS and 1991 for NHSII) and total energy intake. MV-adjusted model was further adjusted for race, highest education level (for NHS and NHSII), highest education level of spouse (NHS), profession (HPFS), census median household income, smoking pack-years, alcohol intake (gram/day), body mass index, high blood pressure, elevated cholesterol, diabetes, stroke, myocardial infarction, angina, coronary artery bypass surgery, cancer, depression, family history of dementia, aspirin use, multivitamin use, and post-menopausal hormone use (in NHS and NHSII). False-discovery-rate corrections were applied across dietary patterns for trend test (P-trends).