





Diet quality and all-cause mortality among US adults, estimated from National Health and Nutrition Examination Survey (NHANES), 2003–2008

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Abstract

Objective: We assessed the ability of the Prime Diet Quality Score (PDQS) to predict mortality in the US population and compared its predictiveness with that of the Healthy Eating Index-2015 (HEI-2015).

Design: PDQS and HEI-2015 scores were derived using two 24-h recalls and converted to quintiles. Mortality data were obtained from the 2015 Public-Use Linked Mortality File. Associations between diet quality and all-cause mortality were evaluated using multivariable Cox proportional hazards models, and predictive performance of the two metrics was compared using a Wald test of equality of coefficients with both scores in a single model. Finally, we evaluated associations between individual metric components and mortality.

Setting: A prospective analysis of the US National Health and Nutrition Examination Survey (NHANES) data.

Participants: Five-thousand five hundred and twenty-five participants from three survey cycles (2003–2008) in the NHANES aged 40 years and over.

Results: Over the 51 248 person-years of follow-up (mean: 9.2 years), 767 deaths were recorded. In multivariable models, hazard ratios between the highest and lowest quintiles of diet quality scores were 0.70 (95% CI 0.51, 0.96, $P_{\text{trend}} = 0.03$) for the PDQS and 0.77 (95% CI 0.57, 1.03, $P_{\text{trend}} = 0.20$) for the HEI-2015. The PDQS and HEI-2015 were similarly good predictors of total mortality ($P_{\text{difference}} = 0.88$).

Conclusion: Among US adults, better diet quality measured by the PDQS was associated with reduced risk of all-cause mortality. Given that the PDQS is simpler to calculate than the HEI-2015, it should be evaluated further for use as a diet quality metric globally.

Keywords

Diet quality metrics
Healthy Eating Index-2015
Prime Diet Quality Score
NHANES
All-cause mortality

Despite a wide palette of dietary indices and scores for measuring various aspects of diet, researchers and policymakers are still searching for a universal tool for describing and tracking the overall quality of diets globally^(1,2). From the global public health perspective, such a metric should be sufficiently simple to use, predictive of health outcomes associated with both undernutrition and overnutrition, applicable to both developed and developing countries, usable across population groups, sensitive enough to track dietary changes over time and, whenever possible, taking into consideration the environmental effects of human diets^(3,4). Having comparable cross-country indicators would also

be beneficial for tracking progress of the UN's Sustainable Development Goals, specifically Goal 2⁽⁵⁾, which calls for 'reducing all forms of malnutrition by 2030'. On a national level, many countries without resources to conduct expensive diet monitoring would benefit from including simple diet quality monitoring tools in their economic or general health surveys⁽⁶⁾.

Currently available diet quality indices may be inappropriate for global use for different reasons. The Healthy Eating Index-2015 (HEI-2015)⁽⁷⁾ or Alternate Healthy Eating Index 2010⁽⁸⁾, for instance, while validated against a range of health outcomes, contain multiple nutrient

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components and require food composition data, making them too complex to calculate in some settings. Other validated metrics such as the Healthy Nordic diet score⁽⁹⁾ or the modified Mediterranean diet score⁽¹⁰⁾ are either population-specific or include alcohol components and may not be appropriate for general use. Further, many diet quality scores were developed primarily for tracking dietary risk of noncommunicable disease, not intake of nutrients of concern in low- and middle-income countries, and therefore may not be suitable for measuring diet quality globally. Finally, diet diversity scores developed for application in low- and middle-income countries, including the Minimum Diet Diversity–Women⁽¹¹⁾ and Food Group Index⁽¹²⁾, while food based and easy to utilise, were not associated with several noncommunicable diseases in previous analyses^(13,14).

In an analysis of FFQ data among US women, the Prime Diet Quality Score (PDQS), a recently developed food-based measure of diet quality, was found to predict CHD, gestational diabetes and hypertension in pregnancy^(13,14). The PDQS was also associated with a lower risk of short telomeres in a Spanish elderly cohort⁽¹⁵⁾, a lower prevalence of individual and clustered cardiovascular risk factors (obesity, hypertension, diabetes and dyslipidaemia among Spanish elderly adults with metabolic syndrome⁽¹⁶⁾ and lower risks of preterm birth, low birth weight and fetal loss among Tanzanian women⁽¹⁷⁾. In light of its simplicity (in that it does not require food composition data and nutrient-level analyses), the PDQS should undergo further validation, particularly with respect to other health outcomes globally, including mortality, and optimisation for use with various types of dietary data, including 24-h recalls, because this is often the data that are available from national surveys. If shown useful in different settings and with respect to different health outcomes, the PDQS could serve as a basis for developing a standalone diet quality assessment tool.

The role of diet quality in mortality has been consistently demonstrated in both the developed and developing world^(18–20). Due to a rapid epidemiological and nutrition transition, evaluation of diet–disease and diet–mortality relationships in high-income countries where large data sets are available, such as the National Health and Nutrition Examination Survey (NHANES), represents an important step in a process of metric evaluation. The HEI-2015 performed well in predicting premature death⁽²¹⁾ in several US cohorts. Although there are not many studies that use NHANES data to examine the association between HEI-2015 and mortality, studies among elderly or metabolically-obese normal-weight persons^(22,23), and a study of the original HEI among adults in NHANES⁽²⁴⁾ suggested inverse associations between diet quality and all-cause mortality.

As part of our wider efforts to develop and comprehensively assess the PDQS in relation to health outcomes and across different populations and economic settings, in the

current study we evaluated the validity of the PDQS against all-cause mortality in NHANES and compared its performance with that of the HEI-2015.

Methods

Study population

The NHANES is a repeated cross-sectional, stratified, multistage probability survey of the US population. Detailed description of the survey design is published elsewhere⁽²⁵⁾. Since 1999, NHANES has been a continuous survey with two years representing one cycle. Members of participating households provide data on diet (via multiple-pass 24-h recall), health and health behaviours and undergo physical examinations at a mobile examination center⁽²⁵⁾. Administration of certain NHANES questionnaires is repeated by telephone 3–10 d after physical examinations. Since 2003, the follow-up assessment included the 24-h recall module as well (collected by telephone); the present analysis includes data from the 2003 to 2004 NHANES and later survey cycles, in order to make use of repeated dietary recalls for the purpose of accounting for within-person variation in statistical analysis. We analysed three consecutive NHANES cycles (2003–2004, 2005–2006 and 2007–2008); 2007–2008 was chosen as the last cycle to allow sufficient exposure lag because 2015 was the latest year from which mortality data were available. Participants younger than 40 years were excluded from the analysis; this cut-off was chosen in order to restrict the sample to participants at a higher risk of dying, while at the same time avoiding major reductions in sample size.

Of 9643 participants with 2 days of dietary intakes in NHANES 2003–2004, we excluded 1289 (13%) participants for whom at least one of two 24-h recalls were flagged as unreliable; 213 (2%) who reported currently being pregnant; 745 (8%) who reported a having history of myocardial infarction, congestive heart failure, stroke or cancer; 280 (3%) with diagnosed diabetes mellitus and 5356 (56%) who were younger than 40 years. For NHANES cycle 2005–2006, of 9950 participants, we excluded (according to the same order and criteria applied in 2003–2004) 1521 (15%), 200 (2%), 617 (6%), 298 (3%) and 5528 (56%) participants, respectively, and in NHANES cycle 2007–2008, of 9762 participants, we excluded 1924 (20%), 39 (1%), 856 (9%), 424 (4%) and 4451 (46%), respectively. The final study sample consisted of 5525 individuals (see online supplementary material, Supplementary Figure 1). The study was exempt from IRB review because it included secondary analysis of deidentified data.

Diet quality scores

We used data from two 24-h diet recalls per person to construct derive the HEI-2015 and PDQS for each individual. The HEI-2015⁽⁷⁾, derived from the USDA Food Patterns, is a measure of diet quality based on adherence to the 2015 Dietary Guidelines for Americans⁽²⁶⁾. It consists of





thirteen components, including three nutrient-based ones (the ratio of polyunsaturated (PUFA+MFA)/SFA, saturated fat and Na) and ten food groups. The maximum HEI-2015 score is 100. To calculate point values for the food-based components, we used the MyPyramid Equivalents Database (MPED 2.0) for the 2003–2004 survey cycle and Food Pattern Equivalents Databases for 2005–2006 and 2007–2008; point values for nutrient-based components were calculated using total nutrient intakes in NHANES. To derive HEI-2015 scores for each individual, we first summed all amounts of dietary constituents over 2 days pertaining to thirteen HEI-2015 components separately, as well as energy amounts corresponding to the same dietary constituents. Then, we calculated thirteen ratios for each individual using these 2-d sums and subsequently scored the ratios according to the HEI scoring standards for each component. The component scores were then summed to calculate the total HEI-2015 score for each individual⁽²⁷⁾.

The PDQS was developed from the PrimeScreen questionnaire⁽²⁸⁾ to serve as a simple-to-use, global diet quality metric. Selection of the score components was based on expert knowledge of diet–disease relationships and involved selecting food groups high in selected dietary constituents (Table 1). The PDQS is a fully food-based score, making any PDQS-based standalone diet assessment tools easy to utilise (without requiring food composition databases and nutrient analyses). Initially, the PDQS consisted of fourteen “healthy” food group components (dark green leafy vegetables, cruciferous vegetables, carrots, other vegetables, citrus fruits, other fruits, legumes, nuts and seeds, poultry, fish, eggs, whole grains, low fat dairy and liquid vegetable oils) and seven “unhealthy” ones (red meat as a main dish, processed meat, potatoes, refined grains and baked goods, sugar-sweetened beverages, fried foods and sweets and ice cream)^(13,14). In the current study, we modified the components (Table 1) to separate deep orange fruits from vegetables (containing >130 RAE/100g)⁽¹²⁾, add deep orange tubers as a positive component and to classify eggs as a ‘neutral’ component among adults, resulting in 15 ‘healthy’, 7 ‘unhealthy’ and 1 ‘neutral’ component. Given that ‘eggs’ remain a PDQS component (for use in low- and middle-income countries and among small children), in the current study, we assigned a point value of ‘2’ to each participant. This approach was already used in our previous analysis⁽²⁹⁾, as a form of ‘neutral’ coding.

Deriving PDQS for NHANES participants consisted of several steps. First, we assigned a PDQS component to each USDA food code in the Food and Nutrient Database for Dietary Studies (FNDDS), using the food code descriptions whenever possible. There were multiple instances (for example, in case of determining whether a food should be categorised as ‘refined grain’ or ‘whole grain’), in which we consulted the FNDDS ingredient nutrient values. Simple foods (such as cabbage, lemon or ice cream) were assigned a single PDQS code.

Composite foods, on the other hand, were assigned two PDQS codes corresponding to their two major component ingredients (based either on the title or the two leading ingredients from the FNDDS ingredient nutrient files), each weighed by 0.5. For example, a chili with beef and beans would be coded both as ‘red meat’ and ‘legumes’. Then, amounts of each food in grams were summed up with other reported foods from each of these food groups. Finally, the total consumed daily amounts of each food group were then compared with thresholds for that PDQS component.

Foods unaccounted for by the PDQS, such as high-fat dairy, seafood or various spices and alcoholic beverages, as well as eggs (while formally a PDQS component, eggs were not scored in the current analysis), were excluded from the analysis. Finally, we calculated the PDQS using two scoring approaches to evaluate its robustness. For approach 1 (Table 1), piloted elsewhere⁽²⁹⁾, a minimum amount was set for foods assigned to each PDQS component that was considered as meeting the requirement. For ‘healthy’ components, we assigned a point value of ‘0’ if no foods from a component were eaten at the minimum amount on either day, a ‘1’ if foods from a component were consumed at the minimum amount on one of the 2 days and a ‘2’ if at least one food from a component was consumed at the minimum amount on both days. ‘Unhealthy’ components were reversely coded (i.e. a ‘0’ was assigned if foods from a component were consumed on both days and a ‘2’ if no foods from a component were consumed on either day). For approach 2 (Table 1), a standard portion size was identified for each food group based on a single food considered broadly representative of each component and converted into three daily gram ranges (corresponding to point values of 0, 1 and 2 for ‘healthy’ components and 2, 1 and 0 for the ‘unhealthy’ ones). These ranges, if extrapolated, corresponded to intake frequencies of 0–1, 2–3 and 4+ per week. The described approach appeared to produce a reasonably widespread of low, medium and high intake ranges in analysis of multiple cross-sectional and cohort data sets from China, India, Mexico and several African countries⁽³⁰⁾. These ranges were then applied to simple averages calculated for each food group in order to make use of both diet recall days. In the current study, the PDQS, in both scoring approaches, has a potential range of 2–46 (with eggs treated as a neutral category, everyone was assigned a ‘2’). We did not adjust the PDQS for total energy, as it consists of a limited number of foods/food groups. Hence, any PDQS-based questionnaire that used to collect primary data on diet quality would not obtain data on total energy intake.

Outcome and covariates’ assessment

To determine mortality status, we used NCHS Public-Use Linked Mortality Files linked until 2015 with a probabilistic matching algorithm to the National Death Index. Participants with mortality status 0 (MORTSTAT = 0) were

**Table 1** Prime Diet Quality Score (PDQS) components, modified version

	Details/examples of foods	Minimum cut-off (g/day) (Approach 1)	Scoring ranges (g/day) (Approach 2)	Scientific rationale for inclusion*
Positively scored components				
Dark green leafy vegetables	Spinach, romaine lettuce, kale, turnip greens, collard, chard, arugula, mustard greens, fresh herbs	50	<10/10–39/>39	Folate, beta-carotene, Fe
Cruciferous vegetables	Broccoli, cauliflower, cabbage, brussels sprouts, kohlrabi, Chinese cabbage	50	<11/11–44/>44	Folate, vitamin C, association with cancer, fibre
Deep orange vegetables (>130RAE†/100g)	Carrot, pumpkin, butternut winter squash (orange varieties)	50	<10/10–39/>39	Folate, beta-carotene content
Other vegetables	Tomato, pepper, cucumber, onion, eggplant, zucchini, beetroot, mushrooms, garlic, summer squash (yellow varieties)	50	<26/26–106/>106	Associations with disease, phytochemicals, fibre
Deep orange fruits (>130RAE†/100g)	Mango, ripe papaya, cantaloupe, apricot	50	<28/28–114/>114	Folate, beta-carotene content
Citrus fruits	Orange, lemon, grapefruit, mandarin, tangerine (whole fruit, not juices)	50	<18/18–74/>74	Vitamin C, folate
Other fruits	Apple, peach, pear, plum, banana, grapes, berries, melon, guava, avocado (whole fruit, not juices)	50	<26/26–106/>106	Associations with disease, phytochemicals, fibre
Deep orange tubers (>130RAE†/100g)	Orange sweet potato	50	<14/14–57/>57	Folate, beta-carotene content
Legumes	Beans, peas, lentils, pulses, legume-based products (tofu, soyamilk) (excludes peanut)	30	<10/10–39/>39	Folate, Zn, protein, Fe, fibre
Nuts and seeds	Includes ground (e.g. peanut) and tree nuts, nut and seed butters/tahini; nut/seed-based spices or other condiments high in protein/unsaturated oils	15	<4/4–16/>16	Fatty acids, Zn, protein, fibre
Poultry	Excludes luncheon meat, and pâté. Includes organs.	30	<12/12–48/>48	Protein, Zn, B ₆ , B ₁₂
Fish	Excludes shellfish	30	<16/16–63/>63	Protein, fatty acids, B ₆ , B ₁₂
Whole grains	Breads, cereals, porridges, noodles and products made of cereal flour (fibre: carbohydrate ≥0.1).	50	<4/4–16/>16	Fibre, carbohydrates
Liquid oils	Olive, rapeseed, sunflower, peanut, maize, sesame, etc. Excludes semisolid oils (e.g. coconut and palm oil)	15	<2/2–7.5/>7.5	Fatty acids, vitamin E, vitamin D
Low fat dairy	Milk, cheese, yogurt, kefir, containing 2% or less fat	30	<35/35–139/>139	Zn, Ca, protein, association with colon cancer
Negatively scored components				
White roots and tubers	White, yellow, red potato, yam (white), cassava, tapioca, white/beige sweet potato.	50	<25/25–100/>100	Low fibre, high starch content, proinflammatory
Red meat	Beef, pork, goat, or lamb/mutton. Includes organs.	30	<12/12–48/>48	High SFA, proinflammatory, association with colon cancer
Processed meat	Sausages, salami, bologna, hot dogs, bacon, pâté, luncheon meat	15	<8/8–31/>31	High Na and SFA content, proinflammatory, association with colon cancer
Refined grains and baked goods	Breads, pan dulce, ready-to-eat breakfast cereals, porridges, noodles and products made of flour containing refined grains only (e.g. white pasta, rice, bread, baked goods) (fibre: carbohydrate <0.1).	30	<3.5/3.5–14/>14	Low fibre, high starch content, proinflammatory
Sugar-sweetened beverages	Soft drinks, energy and sports drinks. Excludes sugar-added fruit nectars, milk or cereal-based sugary drinks, fruit syrups, juices	150	<52/52–207/>207	High sugar content, 'empty calories', proinflammatory
Sweets and ice cream	Candy, chocolate, cake, cookie, sugarcane, ice cream, including homemade ones; sugar, honey, other sugary sweeteners	30	<11/11–45/>45	High sugar content, 'empty calories', proinflammatory
Fried foods	Regardless of where they are obtained/consumed	30	<10/10–40/>40	High SFA, potentially TFA through reheating or use of semisolid fat
Neutral components				
Eggs				Protein, vitamins A, D, B ₁₂

*Only the leading reasons for inclusion are given; there could be other benefits for inclusion of each component not listed here.

†RAE, retinol activity equivalent.



considered to be alive through the end of 2015. Analysis included potential confounders of the association: sex, age (continuous), race/ethnicity (non-Hispanic black, non-Hispanic white, Hispanic, Asian and Native American), days of the week on which the two recalls were recorded (week-end/weekend, weekend/weekday, weekday/weekday, weekday/weekend), smoking status (current, past, never smoker), alcohol consumption (nondrinker, 1 drink/week, 2–4 drinks/week, 5–10 drinks/week, 11–18 drinks/week and 19+ drinks/week, missing), tertiles of physical activity score and BMI (<25, 25–29.9, 30–34.9, 35 and above, missing). The physical activity score was created from the NHANES questionnaire on physical activity over the past 30 d, by assigning one point for each of the reported moderate- or vigorous-level activities and deducting a point for each sedentary activity. We opted for the questionnaire data as physical activity monitor data were not available for one of the three cycles. However, as the physical activity questionnaire was also changed in 2007–2008, we selected the most similar variables in each cycle. The described approach enabled us to order to rank participants by the level of their physical activity in a uniform way across the three survey cycles. Three participants who refused or gave uninformative answers about their smoking status were coded as ‘past smoker’, while 1003 participants with missing alcohol intake and sixty four with missing BMI were coded as ‘missing’, allowing us to keep them in the study sample. The percentage of missing values for each covariate ranged from <1% for smoking to 18% for alcohol intake.

Statistical analysis

We assessed differences in participant characteristics between the top (Q5) *v.* reference (Q1) quintile of diet quality score using χ^2 (for categorical) and Mann Whitney U (for continuous variables) tests. Cox proportional hazards models of time to mortality using time as the metric were used to evaluate the association with the diet quality indices. The assumption of proportional hazards was assessed using appropriate residuals plots (Schoenfeld, scaled Schoenfeld and martingale) and tests of interaction between each variable with time, which revealed no significant departures for either diet score or any covariates. Diet scores were evaluated both as categorical (quintiles) and continuous exposures. Each continuous diet score was categorised into quintiles for use in multivariate Cox regression models (Model 1 adjusted for age, sex and race and Model 2 fully-adjusted for covariates) to evaluate associations between diet quality and mortality over time. Adjusted hazard ratios and their 95% CI were estimated with the lowest quintile of diet quality score (Q1) as the reference group. Because the scores were on different scales and not normally distributed, they were first standardised by conversion to probit scores and then used as continuous exposures (1-SD increase) in Cox regression models. Linear tests for trend were performed by

substituting the observed diet scores for each participant with the mean score within the quintile to which the participant belonged. In a stratified analysis, in a separate set of Cox models, we evaluated interactions between diet quality Z-scores with sex (male *v.* female), age (40–60 *v.* >60 years), BMI (<25 *v.* \geq 25) and smoking status (smoker *v.* non-smoker/past smoker) adjusted for all other covariates. In a third Cox model, we formally compared the performance of the two scores in predicting all-cause mortality by fitting PDQS and HEI-2015 diet quality probit scores in a model and using a Wald test to obtain a p-value for the difference between the PDQS and HEI-2015 β coefficients. Probit scores can be used^(8,14) to standardise to the same scale scores that are not necessarily normally distributed. In a fourth set of Cox models for each score, we evaluated associations between each diet score component and mortality, adjusting for all other score components and covariates. In a sensitivity analysis, due to potential for reverse causality (as some participants might have improved their diets after being diagnosed with an illness), we excluded deaths occurring in the first 2 years of follow-up after the second diet recall in each wave. We accounted for survey-based design of NHANES in the analysis whenever feasible; hence, most results are interpretable as representative of eligible US population. Due to statistical intricacies related to simultaneous adjustment for sampling weights and implementation of the aforementioned Wald test, comparison of predictive powers of the two scores and effect modification analyses (Supplementary Table 3) do not include sampling weights. Statistical analyses were performed using R Statistical Software (Foundation for Statistical Computing, Vienna, Austria) version 3.5.1. All tests were two-sided, with $P < 0.05$ considered statistically significant.

Results

Over 51 248 person-years of follow-up from 5525 individuals (mean: 9.2 years), 767 deaths were recorded. On average, participants in the top quintile of diet score were older, with a higher proportion of females, never smokers and physically active persons and had higher intakes of fibre, vitamins A, B₁, B₂, B₆, C, folate, Ca, Mg, K and Fe, as well as lower intakes of total fat (PDQS only), SFA, Na, carbohydrates and total sugars (PDQS only) compared with the reference diet score quintile (Table 2).

In age, sex and race-adjusted models (Model 1), HR pertaining to overall survival comparing the highest (Q5) *v.* lowest (Q1) quintiles were 0.51 (95% CI 0.39, 0.67; $P_{\text{trend}} < 0.0001$) for the PDQS scoring approach 2, 0.53 (95% CI 0.42, 0.68; $P_{\text{trend}} < 0.0001$) for the PDQS scoring approach 1, and 0.60 (95% CI 0.46, 0.79; $P_{\text{trend}} = 0.0008$) for the HEI-2015 (Table 3). After also adjusting for other covariates (Model 2), the HR somewhat attenuated to


Table 2 Characteristics of the inferred population by quintiles of diet quality scores*

	HEI-2015				PDQS (Scoring approach 1)†				PDQS (Scoring approach 2)‡			
	Q1		Q5		Q1		Q5		Q1		Q5	
	Median	Q1, Q3	Median	Q1, Q3	Median	Q1, Q3	Median	Q1, Q3	Median	Q1, Q3	Median	Q1, Q3
Crude sample (n)§	1069		1128		656		1262		862		1038	
Weighted sample (n)§	18,244,113		18,332,935		11,183,184		21,529,920		14,568,313		18,592,292	
Diet score§	36	33, 40	65	62, 69	10	9, 11	21	20, 22	10	9, 11	21	20, 22
Age (year)§	50	44, 59	54	47, 64¶	50	44, 57	55	48, 65¶	50	44, 59	55	48, 65¶
Male (%)§	54		35¶		62		34¶		59		34¶	
Race/ethnicity§												
White, non-Hispanic (%)	75		80		75		81		74		81	
Black, non-Hispanic (%)	11		8		13		8¶		13		7¶	
Hispanic (%)	8		9		8		7¶		8		7¶	
Smoking§												
Smoker (%)	32		11¶		40		9¶		38		8¶	
Never smoker (%)	42		56¶		38		57¶		40		58¶	
Past smoker (%)	26		33¶		21		34¶		22		34¶	
Alcohol (times/week)§	2	0, 6	1	0, 3¶	2	0, 7	1	0, 3¶	2	0, 6	1	0, 3¶
Physical activity level§,												
Low (%)	38		28¶		39		27¶		40		28¶	
Medium (%)	47		50¶		47		46¶		45		44¶	
High (%)	15		22¶		14		27¶		14		28¶	
BMI (kg/m ²)§	28	24, 33	27	24, 30	28	24, 33	27	24, 31	28	24, 33	27	24, 31¶
Total energy (kJ)§	7933	6184, 10657	7883	6021, 10025	9255	7247, 11811	7899	6096, 9958¶	8627	6594, 11159	7899	6012, 9954¶
Carbohydrate (g)§	218	164, 296	228	179, 298	258	197, 341	226	178, 295¶	242	184, 320	227	175, 292¶
Protein (g)§	74	55, 100	76	58, 98	77	59, 100	80	63, 100	74	55, 96	81	63, 99¶
Total fat (g)§	72	55, 101	70	50, 95	86	64, 117	69	47, 93¶	79	60, 110	67	47, 94¶
SFA (g)§	25	18, 36	21	15, 29¶	29	22, 40	21	14, 29¶	27	20, 38	20	14, 29¶
Fibre (g)§	11	8, 15	19	14, 26¶	11	8, 15	20	15, 26¶	11	8, 15	20	15, 26¶
Total sugars (g)§	85	52, 125	109	83, 147¶	121	86, 175	98	70, 133¶	114	80, 162	98	69, 135¶
Vitamin A§	405	251, 462	724	450, 1030¶	379	249, 583	777	545, 1079¶	369	238, 580	768	544, 1102¶
Vitamin B ₁ §	1.5	1.1, 2	1.6	1.1, 2	1.4	1.1, 1.9	1.6	1.2, 2.1¶	1.4	1, 1.8	1.6	1.2, 2.1¶
Vitamin B ₂ §	1.9	1.5, 2.6	2.2	1.7, 3¶	2	1.4, 2.6	2.3	1.8, 3¶	1.9	1.4, 2.5	2.3	1.7, 3¶
Vitamin B ₆ §	1.4	1, 2	2	1.5, 2.7¶	1.5	1.1, 2.2	2.1	1.6, 2.8¶	1.5	1.1, 2	2.1	1.5, 2.7¶
Vitamin B ₁₂ §	3.8	2.7, 6.1	4.9	3.2, 7.4¶	4.5	2.9, 6.3	4.8	3.3, 7.4	4.1	2.6, 6.2	4.7	3.1, 7.2¶
Vitamin C§	38	18, 79	93	55, 150¶	35	15, 80	102	65, 151¶	38	17, 86	108	65, 155¶
Folate (µg)§	243	326, 466	294	403, 568¶	302	227, 420	445	324, 591¶	291	225, 401	445	325, 593¶
Ca (mg)§	723	502, 961	857	592, 1142¶	692	499, 928	932	720, 1250¶	658	480, 930	944	702, 1281¶
Mg (mg)§	229	176, 300	330	250, 427¶	229	172, 301	335	264, 430¶	221	172, 289	337	265, 433¶
K (mg)§	2208	1700, 2825	3035	2307, 3723¶	2440	1798, 3987	3025	2448, 3720¶	2293	1754, 2972	3046	2470, 3727¶
Fe (mg)§	13	10, 18	15	11, 20¶	13	10, 17	16	11, 21¶	13	9, 17	15	11, 22¶
Zn§	10	7, 14	11	8, 15¶	11	8, 15	11	8, 14	11	8, 15	11	8, 14
Na (g)§	3.4	2.6, 4.5	2.6	2, 3.5¶	3.4	2.6, 4.5	2.9	2.2, 3.9¶	3.1	2.5, 4.3	2.9	2.1, 3.9¶

*Higher scores indicate greater dietary quality. HEI-2015: Healthy Eating Index-2015; PDQS, Prime Diet Quality Score. Q, quintile. N 5525.

†'Healthy' components: '0' = no foods from component eaten during both days, '1' = foods consumed on one day, '2' = foods consumed on both days. 'Unhealthy' components were reversely coded. Minimum quantities used as cut-offs.

‡Daily gram ranges identified using data from several countries, assigning 0, 1 or 2 points for each component (negative components reversely scored) based on the amount consumed during each day.

§Data are presented as weighted medians (Q1, Q3) for continuous variables and weighted percentages for categorical variables to account for survey design.

||Classified into tertiles of a total PA score (a sum of positive points for moderate and intense activities and negative points for sedentary activities).

¶Statistically significant difference ($P < 0.05$) comparing Q1 and Q5, using χ^2 (for categorical variables) and Mann Whitney U (for continuous variables) tests.

**Table 3** Adjusted hazard ratios (HR) (95 % CI) of mortality by quintile of diet quality scores*

	Q1	Q2	Q3	Q4	Q5	<i>P</i> -linear trend	Continuous exposure†
HEI-2015							
Quintile median	36	45	51	57	65		
Quintile range	13–41	41–48	48–54	54–60	60–89		
Model 1‡						0.0008	
HR	1.0 (ref)	0.75	0.74	0.79	0.60		0.84
95 % CI		0.59, 0.95	0.59, 0.94	0.63, 0.98	0.46, 0.79		0.77, 0.92
Model 2§						0.20	
HR	1.0 (ref)	0.78	0.82	0.97	0.77		0.92
95 % CI		0.60, 1.03	0.63, 1.07	0.74, 1.26	0.57, 1.03		0.84, 1.01
PDQS (scoring approach 1) 							
Quintile median	10	14	16	17	21		
Quintile range	5–11	12–14	15–16	17–18	19–29		
Model 1‡						<0.0001	
HR	1.0 (ref)	0.90	0.87	0.66	0.53		0.81
95 % CI		0.70, 1.16	0.65, 1.16	0.49, 0.88	0.42, 0.68		0.75, 0.88
Model 2§						0.01	
HR	1.0 (ref)	1.02	1.07	0.86	0.75		0.91
95 % CI		0.79, 1.32	0.79, 1.45	0.63, 1.19	0.57, 0.99		0.83, 1.00
PDQS (scoring approach 2) 							
Quintile median	10	13	15	17	21		
Quintile range	5–12	12–14	14–16	16–18.5	19–30		
Model 1‡						<0.0001	
HR	1.0 (ref)	0.79	0.78	0.66	0.51		0.81
95 % CI		0.58, 1.06	0.61, 0.99	0.50, 0.87	0.39, 0.67		0.74, 0.88
Model 2§						0.03	
HR	1.0 (ref)	0.83	0.93	0.82	0.70		0.91
95 % CI		0.60, 1.14	0.70, 1.22	0.61, 1.12	0.51, 0.96		0.83, 1.00

PDQS, Prime Diet Quality Score; *N* 5525, events = 767.

*Proportional hazards models used to estimate HR and 95 % CI; higher scores indicate greater dietary quality; HEI-2015: Health Eating Index-2015.

†Standardised to probit scores (1-sd).

‡Models adjusted for age (continuous), sex and race/ethnicity.

§Models adjusted for age (continuous), sex, race/ethnicity, day of week (weekend v. weekday), smoking status (nonsmoker, past and current), alcohol use (nondrinker, 2–4 drinks/week, 5–10 drinks/week, 11–18 drinks/week or 19+ drinks/week), physical activity (low, medium and high), BMI (<25, 25–29.9, 30–34.9, 35 and above).

||'Healthy' components: '0' = no foods from component eaten during both days, '1' = foods consumed on one day, '2' = foods consumed on both days. 'Unhealthy' components were reversely coded. Minimum quantities used as cut-offs.

¶Daily gram ranges identified using data from several countries, assigning 0, 1 or 2 points for each component (negative components reversely scored).

0.70 (95 % CI 0.51, 0.96; $P_{\text{trend}} = 0.03$) for the PDQS scoring approach 2, 0.75 (95 % CI 0.57, 0.99; $P_{\text{trend}} = 0.01$) for the PDQS scoring approach 1, and 0.77 (95 % CI 0.57, 1.03; $P_{\text{trend}} = 0.20$) for the HEI-2015 (Table 3).

When we compared predictive performance of scores against incident mortality, the HEI-2015 was not significantly superior to the PDQS ($P = 0.44$ in Model 1, $P = 0.88$ in Model 2) (Table 4). Among women in fully adjusted models (Supplementary Table 2), the HR were 0.61 (95 % CI 0.42, 0.88; $P_{\text{trend}} = 0.02$) for the PDQS scoring approach 2, 0.76 (95 % CI 0.52, 1.10; $P_{\text{trend}} = 0.03$) for the PDQS scoring approach 1, and 0.60 (95 % CI 0.42, 0.87; $P_{\text{trend}} = 0.03$) for HEI-2015. In men, HR in fully adjusted models were 0.72 (95 % CI 0.51, 1.00; $P_{\text{trend}} = 0.10$) for the PDQS scoring approach 2, 0.75 (95 % CI 0.52, 1.09; $P_{\text{trend}} = 0.11$) for the PDQS scoring approach 1, and 0.85 (95 % CI 0.58, 1.25; $P_{\text{trend}} = 0.58$) for HEI-2015. In interaction analyses by sex, or by age, smoking status and BMI, we found no statistically significant differences in the associations between the PDQS and mortality (Supplementary Table 3). Further, higher scores of the following components of the PDQS were associated with lower mortality: 'other vegetables' (for a 1-point increase, HR = 0.75, 95 % CI 0.63, 0.89), 'nuts and seeds' (HR = 0.82, 95 % CI 0.69,

0.99), 'poultry' (HR = 0.82, 95 % CI 0.67, 0.99), "red unprocessed meat" (HR = 0.85, 95 % CI 0.73, 0.99) and 'sugar-sweetened beverages' (HR = 0.86, 95 % CI 0.74, 0.98). For the HEI-2015 score components, none of the associations were significant (Supplementary Table 4). Finally, in a sensitivity analysis where we removed deaths (*n* 110) that occurred within the first two years after the second diet recall (Supplementary Table 5), HR comparing the highest (Q5) to the lowest (Q1) quintile in fully adjusted models were 0.68 (95 % CI 0.46, 0.98; $P_{\text{trend}} = 0.04$) for the PDQS scoring option 2, 0.71 (95 % CI 0.52, 0.97; $P_{\text{trend}} = 0.01$) for the PDQS scoring option 1, and 0.71 (95 % CI 0.52, 0.98; $P_{\text{trend}} = 0.14$) for HEI-2015.

Discussion

Diet quality, measured by the PDQS, was inversely associated with all-cause mortality among U.S. adults participating in the NHANES between 2003 and 2008, and followed until 2015. In the pooled sample of men and women, HEI-2015 and PDQS had similar inverse associations with total mortality (with the HEI-2015 associations not reaching statistical significance in fully adjusted models). Our results were in line with the

Table 4 Comparing predictive powers of the two diet quality metrics*

	Single score in the model				Both scores in the model				<i>P</i> -difference†
	HEI-2015		PDQS		HEI-2015		PDQS		
	HR	95 % CI	HR	95 % CI	HR	95 % CI	HR	95 % CI	
Model 1‡	0.85	0.79, 0.91	0.82	0.76, 0.89	0.91	0.84, 0.99	0.86	0.79, 0.94	0.44
Model 2§	0.91	0.84, 0.98	0.90	0.83, 0.97	0.94	0.86, 1.02	0.93	0.85, 1.01	0.88

HEI-2015, Healthy Eating Index-2015; PDQS, Prime Diet Quality Score.

*Standardised to probit scores (1-sd).

†*P*-value based on the Wald test evaluating the hypothesis that the β coefficient for the HEI-2015 equals the β coefficient for the PDQS.

‡Adjusted for age (continuous), sex and race/ethnicity.

§Fully adjusted for age (continuous), sex, race/ethnicity, day of week (weekend *v.* weekday), smoking status (nonsmoker, past and current) alcohol use (nondrinker, 2–4 drinks/week, 5–10 drinks/week, 11–18 drinks/week or 19+ drinks/week), physical activity (low, medium, high), BMI (<25, 25–29.9, 30–34.9, 35 and above).

literature on diet quality and mortality as HEI-2010, Alternate Healthy Eating Index, Alternate Mediterranean Diet and Dietary Approaches to Stop Hypertension (DASH) were all associated with a reduced risk of death from any cause in several meta-analyses, and other studies^(10,20,31,32).

The main PDQS findings align with previous analyses of the PDQS documenting inverse associations with a risk of CHD among men and women in several large U.S. cohorts⁽¹³⁾, with a risk of shorter telomeres as markers of cellular aging among elderly Spanish SUN cohort participants⁽¹⁵⁾, and with a lower prevalence of individual and clustered cardiovascular risk factors among elderly participants of the Spanish PREDIMED-Plus cohort⁽¹⁶⁾. While the HR among women appeared somewhat stronger compared with those among men in the HEI-2015 and the PDQS (scoring option 2) models with categorised exposure (Supplementary Table 2), these differences, as well as differences by age, BMI and smoking status were not statistically different in the interaction models, possibly due to lack of power for subgroup analyses. Coming up with a diet score that performs well among both sexes is an important characteristic to strive towards when developing a universal dietary metric. Future analyses should therefore investigate gender-based associations of PDQS with mortality in large cohorts in both developed and developing countries.

Both PDQS scoring approaches led to similar results, indicating the metric robustness and suggesting that selection of appropriate components is more important than the scoring method. However, exploring different component weighting schemes as the next step might still be useful to evaluate nuances in association of the PDQS with under- and over-nutrition-related health outcomes. Having data from multiple days of diet recall has known advantages⁽³³⁾ for evaluating diet-disease relationships, yet a single day per subject is sufficient for tracking mean dietary intakes in a group over time. In addition, dietary data in developing country settings typically include only one diet recall per participant. Therefore, devising a scoring approach that does not require multiple days of diet data (i.e. scoring approach 2) can be of benefit for diet quality tracking purposes.

The evaluation of individual diet score components showed inverse associations of mortality with a higher consumption of 'poultry', 'other vegetables' and 'nuts and

seeds' (PDQS). The association of 'other vegetables' (e.g. tomato, lettuce, pepper, avocado, onion and cucumber) was most probably strong due to the fact that tomato⁽³⁴⁾ and lettuce⁽³⁵⁾ are the most frequently consumed vegetable types. This finding supports consumption of all types of vegetables⁽³⁶⁾. Contrary to the overall literature, higher consumption of red meat and SSB were also associated with a lower mortality in this sample, probably due to chance or reverse causation as the duration of follow-up was not long.

When we formally compared the PDQS's independent predictive ability to that of the HEI-2015, we found that these were similar. This is especially relevant for the needs of global monitoring and tracking of diet quality as PDQS allows for a 'quick and easy' evaluation in settings where food composition databases and expertise for conducting complex dietary surveys may be lacking. In addition, diet assessment tools developed from this diet quality metric could also serve for rapid screening for diet quality and monitoring of patients in health practices. Finally, when we removed the deaths that occurred within the first two years after completing the second diet recall in order to evaluate for potential reverse causality, we observed slightly stronger HR, with the HR in the top quintile of the HEI-2015 reaching statistical significance. This suggests that some participants might have improved their diet quality within two years before death, possibly due to suspicion of illness.

The current study is a part of our aim to develop a universally applicable diet quality metric that can be used across different population groups. Thus, we introduced some variations to the PDQS; for instance, in the present analyses we treated eggs as a 'neutral category' for adults, while it remains a 'positive' component for young children and women in low-income p0countries. We made this decision to accommodate both the findings that while eggs have a minimal overall association with CVD in developed countries, there is a possible positive association among person with diabetes^(37–39) and also that eggs are an important source of protein and choline for women and children in developing countries⁽⁴⁰⁾. Further, we separated 'deep orange fruits' from 'deep orange vegetables' and 'deep orange tubers', to enable separate analyses by these food



components. This scoring could undergo further modifications in the future.

Our study has several strengths. To our knowledge, this is the first analysis of the HEI-2015 and mortality using NHANES data. Second, although the NHANES design is repeated, cross-sectional, mortality data were collected over time, allowing us to do a prospective analysis. Finally, we were able to evaluate and adjust for multiple potential confounding variables. Limitations should also be mentioned. First, a second 24-h recall was not available in NHANES until 2003. Given that at least 2 days of diet recall are required to reduce intra-person variation and calculate PDQS (scoring approach 1), we had to restrict our analysis to three survey waves. This approach inevitably led to reducing the number of cases in the sample. Second, since we used a simple HEI2015 scoring algorithm, we were not able to fully remove the effect of intra-person variation in our scores based on only 2 days of intake; this variation will tend to attenuate association estimates towards the null and may explain non-significant associations with HEI-2015 in fully adjusted models⁽³³⁾. Third, the numbers of deaths in gender-specific analyses were small, and it influenced our ability to detect significant associations in some subgroup analyses. Nevertheless, we were still able to demonstrate clear associations in the overall population, which was the main objective of the current study. And finally, the PDQS models were not adjusted for energy as any PDQS-based questionnaire would not provide data on total energy intake. The unadjusted PDQS therefore better reflects what would be used in practice. However, this could have led to assigning higher scores to persons who tend to eat more of everything and who have a higher chance of being overweight/obese. We did manage to adjust for energy intake to some degree by adjusting for BMI in Model 2, although this may have underestimated the effects of diet quality because some may have been mediated by adiposity. Finally, while there was a disproportionately high proportion of deaths among individuals with missing BMI (38% compared with 15%), there is no reason to believe that the effect of missingness would be differential with respect to the HR estimates related to the two diet scores under examination. Excluding these sixty-four participants did not lead to significantly different HR estimates, but it did slightly reduce the power for tests of statistical significance.

While the PDQS aims to become a universally applicable metric, its food components are to some degree context specific as they may contain different local foods in different settings. Therefore, it will be important to evaluate the validity of this approach in other populations. Future research should also evaluate the PDQS in relation to nutrient adequacy, mortality and other health outcomes in low- and middle-income countries; its use with children; its function as a stand-alone questionnaire to assess diet quality and the extent to which it can evaluate the environmental impacts of food

production systems⁽⁴¹⁾. As an additional next step, it will be important to explore how the PDQS can be integrated into existing national survey platforms. Although most countries do not regularly conduct dietary surveys, household surveys are widely conducted and often include extensive data on household food purchases. These surveys have limitations in nutrition research (stemming in part from the difficulty in capturing intra-household distribution of food and consumption of food away from home). However, observations collected at the household level potentially provide independent information about household food security and a range of household- and individual-level covariates that may help contextualise and add value to diet quality metrics⁽⁴²⁾. It would therefore be worthwhile to develop and evaluate methods for collecting and analysing household measurements to calculate the PDQS and other food-based metrics.

To conclude, we were able to demonstrate that the PDQS is comparable to the HEI-2015 as a predictor of all-cause mortality in a sample of US adults. These findings contribute to the literature on the PDQS, a food-based diet quality metric previously shown to be inversely associated with risks of gestational diabetes, hypertension in pregnancy, CHD, short salivary telomere length and prevalence of cardiovascular individual and clustered risk factors among adults in high-income, and adverse pregnancy outcomes in low-income country settings⁽¹³⁻¹⁶⁾.

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Supplementary material

For supplementary material accompanying this paper visit <https://doi.org/10.1017/S1368980021000859>



References

1. Arimond M & Deitchler M (2019) *Measuring Diet Quality for Women of Reproductive Age in Low- and Middle-Income Countries: Towards New Metrics for Changing Diets*. Washington, DC: Intake – Center for Dietary Assessment/FHI 360.
2. Trijsburg L, Talsma EF, de Vries JHM *et al.* (2019) Diet quality indices for research in low- and middle-income countries: a systematic review. *Nutr Rev* **77**(8), 515–540.
3. Macdiarmid JI, Kyle J, Horgan GW *et al.* (2012) Sustainable diets for the future: can we contribute to reducing greenhouse gas emissions by eating a healthy diet? *Am J Clin Nutr* **96**, 632–639.
4. Macdiarmid JI (2013) Is a healthy diet an environmentally sustainable diet? *Proc Nutr Soc* **72**, 13–20.
5. United Nations (2015) Sustainable Development Goal 2: Zero hunger. <https://www.un.org/sustainabledevelopment/hunger/> (accessed November 2020).
6. Gicevic S, Kremic E, Fung TT *et al.* (2019) Feasibility and sustainability of dietary surveillance, Bosnia and Herzegovina. *Bull World Health Organ* **97**, 349–357.
7. Krebs-Smith SM, Pannucci TE *et al.* (2018) Update of the healthy eating index: HEI-2015. *J Acad Nutr Diet* **118**, 1591–1602.
8. Chiuve SE, Fung TT, Rimm EB *et al.* (2012) Alternative dietary indices both strongly predict risk of chronic disease. *J Nutr* **142**, 1009–1018.
9. Olsen A, Egeberg R, Halkjaer J *et al.* (2011) Healthy aspects of the Nordic diet are related to lower total mortality. *J Nutr* **141**, 639–644.
10. Warensjo Lemming E, Byberg L, Wolk A *et al.* (2018) A comparison between two healthy diet scores, the modified Mediterranean diet score and the Healthy Nordic Food Index, in relation to all-cause and cause-specific mortality. *Br J Nutr* **119**, 836–846.
11. FANTA (2015) *Measuring the Quality of Women's Diets: Consensus on a Global Indicator for Women's Dietary Diversity*. Washington, DC: FHI 360/FANTA.
12. FANTA (2006) *Developing and Validating Simple Indicators of Dietary Quality and Energy Intake of Infants and Young Children in Developing Countries: Summary of Findings from Analysis of 10 Data Sets*. Washington, DC: Food and Nutrition Technical Assistance Project (FANTA), FHI 360.
13. Fung TT, Isanaka S, Hu FB *et al.* (2018) International food group-based diet quality and risk of coronary heart disease in men and women. *Am J Clin Nutr* **107**, 120–129.
14. Gicevic S, Gaskins AJ, Fung TT, *et al.* (2018). Evaluating pre-pregnancy dietary diversity vs. dietary quality scores as predictors of gestational diabetes and hypertensive disorders of pregnancy. *PLoS One* **13**, e0195103.
15. Ojeda-Rodríguez A, Zazpe I, Alonso-Pedrero L *et al.* (2020) Association between diet quality indexes and the risk of short telomeres in an elderly population of the SUN project. *Clin Nutr* **39**(8), 2487–2494.
16. Alvarez-Alvarez I, Toledo E, Lecea O *et al.* (2019) Adherence to a priori dietary indexes and baseline prevalence of cardiovascular risk factors in the PREDIMED-Plus randomised trial. *Eur J Nutr* **59**(3), 1219–1232.
17. Madzorera I, Isanaka S, Wang M *et al.* (2020) Maternal dietary diversity and dietary quality scores in relation to adverse birth outcomes in Tanzanian women. *Am J Clin Nutr* **112**, 695–706.
18. Schwingshackl L, Bogensberger B & Hoffmann G (2018) Diet quality as assessed by the healthy eating index, alternate healthy eating index, dietary approaches to stop hypertension score, and health outcomes: an updated systematic review and meta-analysis of cohort studies. *J Acad Nutr Diet* **118**, 74–100.
19. Collaborators, GBDD (2019) Health effects of dietary risks in 195 countries, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet* **393**, 1958–1972.
20. Mokhtari Z, Sharafkhan M, Poustchi H *et al.* (2019). Adherence to the dietary approaches to stop hypertension (DASH) diet, risk of total, cause-specific mortality: results from the Golestan Cohort study. *Int J Epidemiol* **48**(6), 1824–1838.
21. Panizza CE, Shvetsov YB, Harmon BE *et al.* (2018) Testing the predictive validity of the healthy eating index-2015 in the multi-ethnic cohort: is the score associated with a reduced risk of all-cause and cause-specific mortality? *Nutrients* **10**, 452.
22. Rathod AD, Bharadwaj AS, Badheka AO *et al.* (2012) Healthy eating index and mortality in a nationally representative elderly cohort. *Arch Intern Med* **172**, 275–277.
23. Park YM, Fung TT, Steck SE *et al.* (2016) Diet quality and mortality risk in metabolically obese normal-weight adults. *Mayo Clin Proc* **91**, 1372–1383.
24. Kappeler R, Eichholzer M & Rohrmann S (2013) Meat consumption and diet quality and mortality in NHANES III. *Eur J Clin Nutr* **67**, 598–606.
25. Zipf G, Chiappa M, Porter KS *et al.* (2013) National health and nutrition examination survey: plan and operations, 1999–2010. *Natl Center for Health Statistics Vital Health Stat* **2013**, 1.
26. Agriculture UDoHaHSUDo (2015) *2015–2020 Dietary Guidelines for Americans*. Washington, DC: US Department of Health and Human Services.
27. NCI (2020) Simple HEI scoring algorithm, multiple diet recall days per person, NHANES example. <https://epi.grants.cancer.gov/hei/nhanes-2011-2012-fped-per-person-hei-2015.zip> (accessed November 30 2020).
28. Rifas-Shiman SL, Willett WC, Lobb R *et al.* (2001) PrimeScreen, a brief dietary screening tool: reproducibility and comparability with both a longer food frequency questionnaire and biomarkers. *Public Health Nutr* **4**, 249–254.
29. Gicevic S, Gaskins AJ, Fung TT *et al.* (2019) Demographic and socio-economic predictors of diet quality among adults in Bosnia and Herzegovina. *Public Health Nutr* **22**, 3107–3117.
30. Intake (2020) Global Diet Quality Score (GDQS): a novel food-based metric of diet quality. GDQS Stakeholder Meeting held on September 1, 2020. <https://www.intake.org/news/global-diet-quality-score-gdqs-stakeholder-meeting> (accessed November 2020).
31. Onvani S, Haghghatdoost F, Surkan PJ *et al.* (2017) Adherence to the healthy eating index and alternative healthy eating index dietary patterns and mortality from all causes, cardiovascular disease and cancer: a meta-analysis of observational studies. *J Hum Nutr Diet* **30**, 216–226.
32. Sofi F, Macchi C, Abbate R *et al.* (2014) Mediterranean diet and health status: an updated meta-analysis and a proposal for a literature-based adherence score. *Public Health Nutr* **17**, 2769–2782.
33. Willett WC (2012) *Nutritional Epidemiology*, 3rd ed. New York: Oxford University Press, pp. 101–147.
34. Mazidi M, Katsiki N, George ES *et al.* (2019) Tomato and lycopene consumption is inversely associated with total and cause-specific mortality: a population-based cohort study, on behalf of the International Lipid Expert Panel (ILEP). *Br J Nutr* **22**, 1–21.
35. Liu XM, Liu YJ, Huang Y *et al.* (2017) Dietary total flavonoids intake and risk of mortality from all causes and cardiovascular disease in the general population: a systematic review and meta-analysis of cohort studies. *Mol Nutr Food Res* **61**, 1601003.
36. Wang X, Ouyang Y, Liu J, *et al.* (2014). Fruit and vegetable consumption and mortality from all causes, cardiovascular disease, and cancer: systematic review and dose-response meta-analysis of prospective cohort studies. *BMJ* **349**, g4490.



37. Shin JY, Xun P, Nakamura Y *et al.* (2013) Egg consumption in relation to risk of cardiovascular disease and diabetes: a systematic review and meta-analysis. *Am J Clin Nutr* **98**, 146–159.
38. Djousse L & Gaziano JM (2008) Egg consumption and risk of heart failure in the Physicians' health study. *Circulation* **117**, 512–516.
39. Drouin-Chartier JP, Chen S, Li Y *et al.* (2020) Egg consumption and risk of cardiovascular disease: three large prospective US cohort studies, systematic review, and updated meta-analysis. *BMJ* **368**, m513.
40. Iannotti LL, Lutter CK, Bunn DA *et al.* (2014) Eggs: the uncracked potential for improving maternal and young child nutrition among the world's poor. *Nutr Rev* **72**, 355–368.
41. Willett W, Rockström J, Loken B *et al.* (2019) Food in the Anthropocene: the EAT–Lancet Commission on healthy diets from sustainable food systems. *Lancet* **393**, 447–492.
42. Fiedler JL (2013) Towards overcoming the food consumption information gap: Strengthening household consumption and expenditures surveys for food and nutrition policymaking. *Glob Food Secur* **2**, 56–63.