Diet quality – what is it and does it matter?

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Abstract

Objective: Measures of diet quality have evolved with a number of scoring indices currently in use. They are increasingly being used to examine epidemiological associations between dietary intake and nutrition-related health outcomes. The present review aims to describe current diet quality tools and their applications, and to examine the relationship between diet quality and morbidity and mortality.

Design: A search was conducted of MEDLINE, Cochrane, EMBASE, CINAHL and ProQuest electronic databases. Inclusion criteria were: English language; published from 2004 on; conducted in adult populations; longitudinal/cohort/case–control or cross-sectional study; included a theoretically defined measure of diet quality.

Results: A total of twenty-five indices of overall diet quality and/or variety were found, with components ranging from nutrients only to adherence to recommended food group servings, to variety within healthful food groups. The majority of studies reviewed had methodological weaknesses but demonstrated that higher dietary quality was consistently inversely related to all-cause mortality, with a protective effect of moderate magnitude. The associations were stronger for men and for all-cause and CVD mortality.

Conclusions: The limitations of both the indices and the studies that use them need to be considered when interpreting and comparing results. However, diet quality indices do appear to be able to quantify risk of some health outcomes, including biomarkers of disease and risk of CVD, some cancers and mortality. Further research is needed to improve the validity of these tools and to adapt them for use in clinical dietetic practice.

In recent years, methods for measuring diet quality have evolved and a number of scoring systems or indices to this effect have emerged. This relatively new concept involves the assessment of both quality and variety of the entire diet, enabling examination of associations between whole foods and health status, rather than just nutrients. Diet quality is measured by scoring food patterns in terms of how closely they align with national dietary guidelines and how diverse the variety of healthy choices is within core food groups or equivalent international groupings. More refined scoring methods allow both protective dietary patterns and unfavourable intakes to be identified. As diet quality and variety scores have been examined in association with health outcomes cross-sectionally and to predict such outcomes longitudinally, nutrition interventions could potentially be developed to target improvements in the most critical aspects of an individual's or population's food intake; for example, targeting an increase in fruit and vegetable consumption if an index shows a strong relationship between low intake and CVD.

Kant has previously published two reviews of diet quality indices(1,2), indicating a need for additional validation studies to assess the effectiveness of these indices in predicting nutritional and health status. Thus the aims of the present review were to:

1. Describe current diet quality tools and their applications.
2. Analyse whether higher diet quality scores are associated with lower levels of morbidity and mortality.
3. Examine how robust the studies of association between diet quality and morbidity and mortality are in relation to their findings.

Methods

Search strategy for identification of studies
A systematic review of published English-language literature from 2004 to 2007 was conducted. Relevant literature prior to this time was identified from the reviews previously published by Kant(1,2).

The following electronic databases were searched: MEDLINE, Cochrane, EMBASE (Excerpta Medica Database), CINAHL (Cumulative Index to Nursing and Allied Health Literature) and ProQuest. Scopus was used to

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identify studies that had cited Kant’s 2004 review(2). The MeSH (Medical Subject Headings of the National Library of Medicine) keyword search terms included diet, dietary, quality, variety, diversity, pattern, score, indicator, index, guideline, Healthy Eating Index, Alternative Healthy Eating Index, Recommended Food Score, chronic disease, cancer, cardiovascular disease; and variations of these.

Selection of studies
The current review used specific selection criteria for retrieval of studies. Inclusion criteria were: (i) studies of a longitudinal/cohort, case-control or cross-sectional nature; (ii) human subjects; (iii) adults; (iv) English language; (v) literature published from 2004 onwards; and (vi) use of theoretically defined dietary patterns (food indices/scores) or a measure of diet quality created a priori and based on current nutrition knowledge(3). Pertinent literature published in one of Kant’s previous reviews(2) was adapted and cited to facilitate the aim of the current review.

Specific exclusion criteria included: (i) studies of non-English language; (ii) studies conducted with children or pregnant women; (iii) intervention studies; (iv) published prior to 2004, with the exception of Kant(1,2); (v) studies on animals; and (vi) studies that primarily use dietary patterns derived a posteriori from food consumption data, based and defined empirically or statistically, such as with cluster analysis and factor analysis(3).

Articles were retrieved from the electronic search if information contained in the title, descriptor/MeSH headings and abstract appeared to be consistent with the inclusion criteria. The methodological quality of the articles was critically appraised and a summary was tabulated based on JBI (Joanna Briggs Institute) observational critical appraisal criteria of a random sample, clear inclusion criteria, objective assessment of outcomes, sufficient description of group comparison and appropriate statistical analyses; while study details, key findings and risk reduction ratios were extracted(4,5) and tabulated.

Results
After retrieval of articles, nine articles were selected. These studies were combined with nineteen studies, previously reviewed by Kant(1,2), giving a total of twenty-eight studies included. Table 1 describes the diet quality indices or tools used. Table 2 summarises the quality assessment. Tables 3 and 4 summarise the main features of studies validating the indices cross-sectionally or in a case–control design with biomarker or health outcomes, including the dietary assessment measure, population, main results and study limitations.

Diet quality indices and scores
A total of twenty-five indices of overall diet quality and/or variety were identified (Table 1). The major indices include the Healthy Eating Index (HEI)(5), the Healthy Diet Indicator (HDI)(7), the Healthy Food Index (HFI)(8), the Recommended Food Score (RFS)(9), the Diet Quality Index (DQI)(10), the Diet Quality Score (DQS)(11) and the Mediterranean Diet Score (MDS)(12). Table 1 describes each index, the variations or modifications derived from them and validation studies(6,13–15). The Diet Quality Index International (DQI-I)(16) was not included because rather than examine associations with disease outcomes it compared diet quality between countries.

The majority of indices are based on national nutrition recommendations and national dietary guidelines specific to the country where the tool was developed. Adherence to these recommendations is assessed by the diet quality/variety score and then commonly compared with nutrient intakes (not reported here) and the risk for various health outcomes, including biomarkers of disease, mortality and chronic diseases such as CVD and cancer (Table 2). While most indices have been created for use with the US population, indices based on the Mediterranean diet have emerged due to recent research highlighting associations with reduced risk of CVD and some types of cancer(3,5,12,17–20).

Kant highlighted in 1996(17) that the construction of diet quality indices has taken three major approaches: (i) based on food groups or specific foods; (ii) based on nutrient intakes; or (iii) derived from combinations of foods and nutrient intakes. In 2007 Waijers et al. added that current scores were based on adherence to established national dietary guidelines or a Mediterranean pattern(3). Depending on inclusion or exclusion of specific foods and/or nutrients, diet quality indices can be generated to reflect a dietary intake that is healthy, unhealthy or a combination of both(3). Most indices, including the HEI and DQI, are based on both food groups and nutrients, while some, such as the HFI, are based on foods and food groups (Table 1). The food items and groups selected as components of the indices include vegetables, fruits, cereals and grains, meat products and dairy products, with some specifically including fish and olive oil. Common nutrient components used in scores include total fat, ratios of fat types (ratio of saturated fat to mono- or polyunsaturated fat) and cholesterol. Alcoholic beverages are used in many indices, predominantly Mediterranean indices, in recognition of the proposed beneficial effects and to evaluate associations with health outcomes(3). Other nutrients used include Na, dietary fibre, protein and complex carbohydrates(3).

Dietary variety or diversity is included in some indices (HEI, HEI-f, DQI-R, total and specific food group diversity and its variations), in addition to foods and nutrients, with higher scores awarded for a more varied diet(5). It has been proposed that variety within food groups may be considered a better indicator of more healthful outcomes(3,13,15,21,25–27); however, this is not considered important by some authors(5).
<table>
<thead>
<tr>
<th>Index</th>
<th>Objective</th>
<th>Index method</th>
<th>Dietary method</th>
<th>Study and reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy Eating Index (HEI)</td>
<td>Single, summary measure of diet quality based on nutrients and foods. Assesses adherence to US Food Guide Pyramid and Dietary Guidelines for Americans</td>
<td>Ten components based on aspects of a healthy diet: each contributes 0–10 points. Score range: 0 (worst)–100 (best). Components 1–5 based on conforming to serving recommendations of the US Food Guide Pyramid for five major groups including grains; vegetables; fruit; meat; milk. Others based on overall fat % energy; saturated fat % energy; cholesterol; Na; variety in diet</td>
<td>24 h recall and 2 d food record</td>
<td>Hann et al. (2001)(^{(9)}); Weinstein et al. (2004)(^{(13)}); Fung et al. (2006)(^{(21)})</td>
</tr>
<tr>
<td>Alternative Healthy Eating Index (AHEI)</td>
<td>Assesses whether AHEI predicts disease risk better than HEI. In contrast to HEI it acknowledges benefits of unsaturated oils, distinguishes quality within food groups, and excludes potato and its products from vegetable group</td>
<td>Nine components including vegetables; fruit; nuts and soya; ratio of white to red meat; cereal fibre; trans fat % energy; ratio of polyunsaturated fat to saturated fat; alcohol servings daily; duration of multivitamin use. Eight of nine components contribute 0–10 points each, with 10 indicating recommendations met and 0 indicating the least healthy dietary behaviour. Intermediate intakes scored proportionally between 0 and 10. Component scores summed for AHEI total ranging from 2-5 (worst) to 87-5 (best)</td>
<td>FFQ (-130 item)</td>
<td>McCullough et al. (2002)(^{(22)}); Fung et al. (2006)(^{(21)})</td>
</tr>
<tr>
<td>Healthy Eating Index from Food Frequency Score (HEI-f)</td>
<td>Assesses whether high HEI score, calculated from validated FFQ and Healthy Eating Index Final Report guidelines, predicts lower chronic disease risk</td>
<td>Similar to original HEI. Ten components based on aspects of a healthy diet: each contributes 0–10 points. Score range: 0 (worst)–100 (best). Components 1–5 based on conforming to serving recommendations of US Food Guide Pyramid for five major groups including grains; vegetables; fruit; meat; milk. Others based on overall fat % energy; saturated fat % energy; cholesterol; Na; variety in diet. Variety component varies from original HEI, changing calculation method from number of unique foods consumed in 3 d to number of unique foods consumed at least once monthly</td>
<td>FFQ</td>
<td>McCullough et al. (2000)(^{(23)}); McCullough et al. (2000)(^{(24)})</td>
</tr>
<tr>
<td>Healthy Diet Indicator (HDI)</td>
<td>Based on WHO dietary recommendations for preventing chronic disease</td>
<td>Dichotomous variables used with 1 indicating being within recommendations and 0 being outside recommendations. HDI = sum of variables with range from 0 to 9 (9 as highest value). Food groups include saturated fat; polyunsaturated fat; protein; complex carbohydrates; dietary fibre; legumes/nuts/seeds; fruit/vegetable; mono- and disaccharides; cholesterol</td>
<td>Diet history</td>
<td>Huijbregts et al. (1997)(^{(7)}); Huijbregts et al. (1998)(^{(29)}); Knoops et al. (2006)(^{(20)})</td>
</tr>
<tr>
<td>Healthy Food Index (HFI)</td>
<td>Based on previous diet quality indices and current recommendations for a healthy diet(^{(1)}). Assesses food intake patterns defined a priori on basis of food recommendations and a posteriori by factor analysis, using mortality as the outcome</td>
<td>Four components, each receiving 1 point if met daily: not consuming margarine, butter or lard; consumption of boiled or raw vegetables at least once; consumption of coarse rye or white bread at least once; consumption of fruit at least once. Score range 0–4. Score of 4 indicates better diet quality</td>
<td>FFQ (28 items)</td>
<td>Osler et al. (2001)(^{(8)}); Osler et al. 2002(^{(30)})</td>
</tr>
<tr>
<td>Healthy Food and Nutrient Index (HFNI)</td>
<td>Based on dietary guidelines issued by the National Nutrition Council Belgium</td>
<td>Eight components based on dietary guidelines. Components include saturated fat; monounsaturated fat; polyunsaturated fat; protein; dietary fibre; fruit/vegetables; carbohydrates; dietary cholesterol. 1 point awarded if consumption is within recommendation limits and 0 is awarded if consumption exceeds limit. Score range 0–8, with higher score indicating adherence to recommendations. HFNI expressed in quartiles with 1 being best quartile</td>
<td>1 d food record</td>
<td>Bazelmans et al. (2006)(^{(31)})</td>
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<tr>
<td>Index Method</td>
<td>Objective</td>
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<td>Dietary Method</td>
<td>Study and Reference</td>
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<tr>
<td><strong>Recommended Food Score (RFS)</strong></td>
<td>Based on consumption of recommended foods in current US dietary guidance</td>
<td>Sum of food items (maximum 23) consumed within a week. Dichotomous variables each scoring 1 (for meeting recommendation) or 0 (not meeting the recommendation), with score range of 0–23 (23 highest score)</td>
<td>FFQ</td>
<td>Kant et al. (2000); McCullough et al. (2002); Mai et al. (2005)</td>
</tr>
<tr>
<td><strong>Recommended Food Score (RFS)</strong></td>
<td>Based on ‘good’ foods (consumption of foods from the FFQ list consistent with national dietary guidelines)</td>
<td>Foods consumed 1–3 times monthly score 1 point (maximum of 17). Score range 0–17, with 17 being best score. Differs slightly from previous RFS with poultry, potato and juices excluded</td>
<td>FFQ</td>
<td>Michel and Wolk (2002); Fung et al. (2006)</td>
</tr>
<tr>
<td><strong>Not Recommended Food Score (NRFS)</strong></td>
<td>Based on ‘bad’ foods (consumption of foods not recommended by current dietary guidance)</td>
<td>Foods consumed 1–3 times monthly score 1 point (maximum of 21). Score range 0–21, with 21 being best score. Some foods included: meat; chips; butter; white bread; cheese. Poultry, potato and juices were excluded</td>
<td>FFQ</td>
<td>Michel and Wolk (2002)</td>
</tr>
<tr>
<td><strong>Diet Quality Index (DQI) (1)</strong></td>
<td>Measures quality of diet that can reflect the risk of diet-related chronic disease. Based on nutrients</td>
<td>Based on eight National Research Council Diet and Health recommendations. Includes six nutrient intakes: total fat; saturated fat; cholesterol; protein; Ca; Na; servings from two food groups: vegetables and fruit, grains. Each component scores 0 (meets recommendation), 1 (recommendation almost met) or 2 (recommendation not met). Score range is 0–16, 0 indicates excellent diet</td>
<td>24 h recall and 2 d food record</td>
<td>Seymour et al. (2003)</td>
</tr>
<tr>
<td><strong>Diet Quality Index Revised (DQI-R)</strong></td>
<td>Improved DQI to reflect current dietary guidelines, incorporate dietary moderation and variety, and improve methods of estimating food servings</td>
<td>Ten components instead of eight: each scoring 0–10 points. Score range: 0–100. In contrast to DQI, higher scores indicate adherence to dietary guidance. Since modification, fruit and vegetables are separated according to Pyramid recommendations; includes Fe intake; excludes protein intake; and scores dietary moderation and diversity</td>
<td>24 h recall</td>
<td>Fung et al. (2006)</td>
</tr>
<tr>
<td><strong>Diet Quality Index (DQI) (2)</strong></td>
<td>Measures quality of diet in a Mediterranean population, and reflects risk of diet-related chronic disease</td>
<td>Similar to previous DQI but modified for Mediterranean population. Now only seven components: saturated fat; cholesterol; olive oil; fish; meat; cereals; vegetables; fruit. Each component scores 0 (meets recommendation), 1 (recommendation almost met) or 2 (recommendation not met). Score range is 0–14; a score of 0 indicates an excellent diet</td>
<td>FFQ (162 items)</td>
<td>Gerber et al. (2000)</td>
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<tr>
<td><strong>Diet Quality Score (DQS) (1)</strong></td>
<td>Uses estimated average requirement recommendations of US Dietary Reference Intakes (2000) to define compliance</td>
<td>Seventeen nutrient categories based on age- and gender-specific recommendations. 1 point awarded for recommendation being met and 0 if not met. Score range is 0–17, with 17 indicating highest compliance. Dietary supplements were excluded</td>
<td>24 h recall</td>
<td>Fitzgerald et al. (2002)</td>
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<tr>
<td><strong>Diet Quality Score (DQS) (2)</strong></td>
<td>A crude index of overall quality of diet habits. Based on previous indices of overall diet quality as well as Danish Dietary Guidelines (minimum 600 g vegetables/fruit each day, minimum 200 g fish per week and low fat intake (total fat &lt;30 % energy intake, saturated fat &lt;10 % energy intake))</td>
<td>Three-point score developed for four food groups: fish; vegetables; fruit; fats. Recommended intakes of all groups except fats used to calculate upper cut-off points. Fats group used no use of spread or fat for cooking as a cut-off point for its high score. Lower cut-off points defined by using saturated fats; no consumption of fish; or low intake of vegetables and fruit. Participants categorised into three groups for each food group: very unhealthy (1 point), average intake (2 points) and very healthy (3 points). Points totalled for range of 1–12, 12 being most healthy</td>
<td>FFQ (48-item validated by a 198-item)</td>
<td>Toft et al. (2007)</td>
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<tr>
<td>Index</td>
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<td>Index method</td>
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<td><strong>Dietary Quality (DQ)</strong></td>
<td>Assesses diet quality (2/3 of 1989 Recommended Dietary Allowance) and its association with survival. Nutrient-based</td>
<td>Based on number of nutrient intakes that fall below 67% of the 1989 RDA. Eight nutrients analysed: Fe; protein; Ca; vitamin A; vitamin C; thiamine; riboflavin; preformed niacin. When five or more nutrients were below 2/3 of RDA diet was classed as poor quality. Energy intake, food servings per day and supplement use (not quantified) were also measured.</td>
<td>24 h recall</td>
<td>Murphy et al. (1996)</td>
</tr>
<tr>
<td><strong>Dietary Guidelines Index (DGI)</strong></td>
<td>Measures compliance with 5th edition of Dietary Guidelines for Americans. Food-based; nutrient guidelines excluded. Allows individuals to be ranked according to compliance</td>
<td>Each guideline contributes equally to DGI. Made of nine components each with maximum of 2 points. Score range 0–18 with 18 indicating full compliance with guidelines and 0 being non-compliant. Includes diet-related and non diet-related recommendations.</td>
<td>FFQ (127 items)</td>
<td>Harnack et al. (2002)</td>
</tr>
<tr>
<td><strong>Mediterranean Diet Score (MDS) (1)</strong></td>
<td>Assesses overall diet pattern and survival based on traditional Mediterranean diet</td>
<td>Eight desirable components including: high monounsaturated to saturated fat ratio; high legume consumption; high vegetable consumption; high fruit consumption; high cereal consumption; moderate ethanol consumption; low milk/dairy consumption; low meat/meat product consumption. Cut-off points were used for each component based on median values for each sex. 1 point for each component met, score range 0–8. Higher scores indicate better diet</td>
<td>FFQ (1980 items)</td>
<td>Trichopoulou et al. (1995)</td>
</tr>
<tr>
<td><strong>Mediterranean Diet Score (MDS) (2)</strong></td>
<td>Assesses overall diet pattern based on traditional Mediterranean diet</td>
<td>Similar to previous MDS; however fish added as a component making a total of nine. Beneficial components (legumes, fruit/nuts, vegetables, cereals and fish) received score of 1 if above the sex-specific median cut-off, and 0 if below median. For detrimental components (dairy, meat and poultry), a score of 1 received if below median and 0 if above. In ethanol component, 1 point received for men who consume 10–50 g/d and for women who consume 5–25 g/d. The last component is ratio of monounsaturated fat to saturated fat. Score range is 0–9, with 9 indicating maximal adherence to traditional Mediterranean diet</td>
<td>FFQ (150 items)</td>
<td>Trichopoulou et al. (2003); Lagiou et al. (2006)</td>
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<tr>
<td><strong>Mediterranean Diet Score (MDS) (3)</strong></td>
<td>Assesses association between a Mediterranean diet score and survival in an elderly Danish population; and diet score and biochemical dietary measures</td>
<td>Similar to previous MDS, only seven components as high legume consumption was omitted, and starchy roots were placed in vegetable group not the cereals as above. Score range 0–7, with 7 indicating better diet</td>
<td>3 d food record and frequency checklist</td>
<td>Osler and Schroll (1997)</td>
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<tr>
<td><strong>Mediterranean Diet Score (MDS) (4)</strong></td>
<td>Assesses overall diet pattern based on traditional Mediterranean diet</td>
<td>Similar to original MDS but has nine components. The legumes group was replaced with legumes/nuts/seeds; the vegetable group replaced with vegetable and potatoes; and the meat and meat products group replaced with meat and poultry. Fish was also added as a group. Score range from 0 to 9, with 9 indicating a higher quality diet</td>
<td>Diet history</td>
<td>Knoops et al. (2006)</td>
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<tr>
<td><strong>Mediterranean Adequacy Index (MAI)</strong></td>
<td>Assesses how close the population’s food intake meets reference dietary pattern. This version is varied slightly with respect to the original</td>
<td>The sum of Mediterranean food groups divided by the sum of non-Mediterranean food groups. Mediterranean foods include cereals; legumes; vegetables; fruit; potatoes; fish; monounsaturated fat; wine. Non-Mediterranean foods include milk and its products; meat and poultry; eggs; sugar; saturated fat. Original version used vegetable oil instead of monounsaturated, and animal fats and margarine instead of saturated fat. In the original version food groups are expressed as percentage of total daily intake of energy; however, this was changed to adjusted daily intakes for men and women</td>
<td>Diet history</td>
<td>Knoops et al. (2006)</td>
</tr>
<tr>
<td>Index</td>
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<tr>
<td>Alternative Mediterranean Diet Score (aMed)</td>
<td>Assesses overall diet pattern based on traditional Mediterranean diet</td>
<td>Variation of original MDS. Components have been modified: potato products excluded from vegetable group; fruits and nuts separated into two groups; dairy group eliminated; includes whole-grain products only; includes only red and processed meats for the meat group; and assigning 1 point for 5 and 15 g/d alcohol intake. Scoring system as MDS&lt;sup&gt;(4)&lt;/sup&gt;. The score range is 0–9, with 9 indicating better diet</td>
<td>FFQ (61 expanded to 116 items)</td>
<td>Fung et al. (2006)&lt;sup&gt;(21)&lt;/sup&gt;</td>
</tr>
<tr>
<td>Total and specific food group diversity (1)</td>
<td>Assesses variety of food intake</td>
<td>Based on total number of foods consumed at least once weekly. Quartiles of total diversity and specific food group diversity formed based on distribution of controls including age and sex. Highest quartile indicates more diversity in the diet. Food groups included: vegetables; fruit; meat; carbohydrate; other food</td>
<td>FFQ (29 items)</td>
<td>Fernandez et al. (1996)&lt;sup&gt;(26)&lt;/sup&gt;</td>
</tr>
<tr>
<td>Total and specific food group diversity, and diet composition</td>
<td>Assesses variety of food intake and composition of diet (proportion of food categories)</td>
<td>Diet diversity defined by number of unique foods reported in past year, in addition to diversity within six food groups. Six groups include: meat/fish/poultry/eggs; fruits; vegetables; whole grains; refined grains; dairy foods. Composition defined by estimated proportion of total food items reported in each food group, as well as ratio of plant to animal products. Gender-specific quintiles were produced for diet diversity and composition in the control group. Did not specify calculation of score</td>
<td>Diet history</td>
<td>Slattery et al. (1997)&lt;sup&gt;(27)&lt;/sup&gt;</td>
</tr>
<tr>
<td>Total and specific food group diversity (2)</td>
<td>Assesses variety of food intake</td>
<td>Based on total number of foods consumed at least once weekly. Quartiles of total diversity and specific food group diversity formed based on distribution of controls including age and sex. Highest quartile indicates more diversity in the diet. Food groups included: dairy; bread and cereal; meat; vegetables; fruit</td>
<td>FFQ (79 items)</td>
<td>Fernandez et al. (2000)&lt;sup&gt;(25)&lt;/sup&gt;</td>
</tr>
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</table>
Table 2 Quality assessment of included studies

<table>
<thead>
<tr>
<th>Study and reference</th>
<th>Was the study based on a random or pseudo-random sample?</th>
<th>Were inclusion criteria for the sample clearly defined?</th>
<th>Were outcomes assessed objectively?</th>
<th>Were group descriptions sufficient for comparisons?</th>
<th>Was the statistical analysis appropriate?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trichopoulou et al. (1995)</td>
<td>U</td>
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<td>N</td>
<td>N</td>
<td>CPH; rate ratios</td>
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<td>Y</td>
<td>U</td>
<td>Y</td>
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<td>N</td>
<td>Y</td>
<td>N</td>
<td>CPH</td>
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<td>Y</td>
<td>Y</td>
<td>CPH</td>
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<tr>
<td>Osler and Scroll (1997)</td>
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<td>N</td>
<td>Y</td>
<td>N</td>
<td>CPH; rate ratio</td>
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<td>Y</td>
<td>Y</td>
<td>OR from unconditional multiple logistic regression models</td>
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<td>OR from unconditional multiple logistic regression</td>
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<td>Y</td>
<td>N</td>
<td>Wilcoxon signed rank test; Spearman rank correlations</td>
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<td>CPH</td>
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<td>N</td>
<td>Y</td>
<td>N</td>
<td>RR from pooled logistic regression</td>
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<tr>
<td>McCullough et al. (2000)</td>
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<td>Y</td>
<td>RR from pooled logistic regression</td>
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<td>Harn et al. (2003)</td>
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<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Pearson correlations; stepwise regression</td>
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<td>Y</td>
<td>U</td>
<td>CPH; hazard rate ratio</td>
</tr>
<tr>
<td>Fitzgerald et al. (2002)</td>
<td>U – previously published</td>
<td>U</td>
<td>Y</td>
<td>Y</td>
<td>OR from logistic regression</td>
</tr>
<tr>
<td>Harnack et al. (2002)</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>CPH; relative risk</td>
</tr>
<tr>
<td>McCullough et al. (2002)</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>OR from logistic regression</td>
</tr>
<tr>
<td>Michels and Wolk (2002)</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
<td>CPH; hazard ratios</td>
</tr>
<tr>
<td>Osler et al. (2002)</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>CPH; hazard rate ratio</td>
</tr>
<tr>
<td>Seymour et al. (2003)</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
<td>CPH; hazard rate ratio</td>
</tr>
<tr>
<td>Huijbregts et al. (2003)</td>
<td>U</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>CPH; hazard rate ratio</td>
</tr>
<tr>
<td>Weinstein et al. (2004)</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>Weighted Pearson correlations; multiple linear regression</td>
</tr>
<tr>
<td>Mai et al. (2005)</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>CPH; relative risk</td>
</tr>
<tr>
<td>Bazelmans et al. (2006)</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>Mann–Whitney tests; t tests; Pearson’s (x^2) tests; logistic regression</td>
</tr>
<tr>
<td>Fung et al. (2006)</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>Y</td>
<td>CPH; relative risk</td>
</tr>
<tr>
<td>Knoops et al. (2006)</td>
<td>U – previously published</td>
<td>U</td>
<td>Y and N</td>
<td>Y</td>
<td>CPH; hazard rate ratio</td>
</tr>
<tr>
<td>Lagiou et al. (2006)</td>
<td>Y</td>
<td>U</td>
<td>Y</td>
<td>Y</td>
<td>CPH; hazard rate ratio</td>
</tr>
<tr>
<td>Toft et al. (2007)</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>Spearman’s correlations; ordinal logistic regression; linear and multiple regression</td>
</tr>
<tr>
<td>Total</td>
<td>9Y, 7U, 12N</td>
<td>12Y, 7U, 9N</td>
<td>23/24Y, 1U, 34N</td>
<td>14Y, 3U, 11N</td>
<td>28Y</td>
</tr>
</tbody>
</table>

U, unclear; N, no; Y, yes; CPH, Cox proportional hazard model.

*All studies Yes for statistical tests.
<table>
<thead>
<tr>
<th>Study and reference</th>
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<th>Subjects</th>
<th>Main outcome</th>
<th>Results</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Huijbregts <em>et al.</em> (1998)</td>
<td>HDI</td>
<td>Diet history</td>
<td>1049 men aged 70–91 years. Seven Countries Study: Finland, Italy and The Netherlands</td>
<td>Cognitive function. Measured by MMSE</td>
<td>One of five cohorts suggested higher HDI = statistically significant lower prevalence of cognitive impairment. Variation in prevalence of cognitive impairment: 14.4 % in The Netherlands to 42.1 % in one cohort in Italy</td>
<td>Cross-sectional design with dietary data collected retrospectively after 30-year follow-up. Variation in cohort participation rates. Possible differences in precision of dietary reporting associated with cognitive function</td>
</tr>
<tr>
<td>Gerber <em>et al.</em> (2000)</td>
<td>DQI (2)</td>
<td>162-item FFQ</td>
<td>146 adults aged 22–75 years. South France</td>
<td>Plasma carotenoid and vitamin E; plasma TAG and cholesterol; fatty acids in erythrocyte membranes</td>
<td>Poor diets had lower EPA, DHA, vitamin E and β-carotene concentrations and higher cholesterol concentrations. DQI significantly correlated only with EPA and DHA; vitamin E borderline significant. After adjustment for smoking, DQI significantly correlated with EPA, DHA, vitamin E and β-carotene</td>
<td>Items and score limits used in DQI limit application of tool on various populations. Fat recommendation (&lt;30 %) not suitable for population studied. Cross-sectional design of the study. Small cohort</td>
</tr>
<tr>
<td>Hann <em>et al.</em> (2001)</td>
<td>HEI</td>
<td>3 d food record</td>
<td>340 women aged 21–80 years. USA</td>
<td>Carotenoids, vitamin C, folate and cholesterol</td>
<td>HEI correlated with plasma carotenoids (except lycopene), folate and vitamin C level</td>
<td>Possible confounding factors include: income level, education level, economics of food choice. Cross-sectional design of the study. Small cohort</td>
</tr>
<tr>
<td>Weinstein <em>et al.</em> (2004)</td>
<td>HEI</td>
<td>24 h recall</td>
<td>16 467 adults aged ≥17 years. NHANES III, USA</td>
<td>Serum vitamins A, B₁₂, C, D and E; RBC and serum folate; serum carotenoids, ferritin, Se, TAG, homocysteine and cholesterol</td>
<td>HEI had positive correlation with RBC and serum folate, serum vitamin C and E, and serum carotenoids (except lycopene). Results were 21–175 % higher in highest HEI group. Strongest associations occurred in biomarkers of vegetable and fruit consumption. Mean HEI significantly higher in those who consumed dietary supplements (42 % of participants). Partial correlations became attenuated once compared with crude correlations</td>
<td>Biomarkers used were limited as represent nutrients found in vegetables and fruit, therefore does not reflect whole diet. Use of 24 h recall as dietary measure. Possible confounding due to supplement use. Cross-sectional design of the study.</td>
</tr>
<tr>
<td>Toft <em>et al.</em> (2007)</td>
<td>DQS (2)</td>
<td>48-item FFQ (validated by 198-item FFQ)</td>
<td>6542 men and women aged 30–60 years. Inter99 study, Denmark</td>
<td>Total cholesterol, TAG, HDL-C, LDL-C, homocysteine and blood pressure Risk of IHD estimated by Copenhagen Risk Score</td>
<td>DQS had negative association with total cholesterol, LDL-C, TAG, homocysteine and absolute risk of IHD (adjusted for age, sex, physical activity level and smoking). DQS had positive association with HDL-C</td>
<td>Similarity in two FFQ used may have caused overestimation of score validity; however, different time period measures in two FFQ may have caused underestimation of score validity. Cross-sectional design of the study</td>
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</tbody>
</table>

NHANES III, Third National Health and Nutrition Survey; MMSE, Mini-Mental State Examination; RBC, red blood cell; HDL-C, HDL cholesterol; LDL-C, LDL cholesterol.
Scoring methods and cut-off points to assess diet quality vary across indices. The various MDS indices use sex-specific median cut-off points\(^{(12,17–21)}\). Median cut-off points allow subjects to be scored positively or negatively for each item\(^{(3)}\). Indices such as the Dietary Guidelines Index (DGI)\(^{(28)}\) use lower, intermediate and upper cut-off levels\(^{(3)}\), while the HDI uses dichotomous values to determine whether dietary recommendations have been met or not. For example, with the HDI, if a person consumed 27–40 g dietary fibre daily, he/she was awarded 1 point for the recommendation being met\(^{(3)}\). It is debatable whether cut-off boundaries, dichotomous values or continuous variables allow for a better evaluation of adherence to recommendations or examination of associations with disease outcomes.

**Health outcomes associated with diet quality indices**

Diet quality indices can also be used to measure risk of various health outcomes, including biomarkers of disease (Table 3), mortality and chronic diseases (Tables 4 and 5). These are discussed by major dietary index type to allow better comparison of the study results.

**Healthy Eating Index**

The HEI was used in three studies\(^{(6,13,21)}\) and was associated with biomarkers such as carotenoids (except lycopene), cholesterol and vitamins C and E\(^{(6,13)}\). The strongest associations occurred for the biomarkers of vegetable and fruit consumption\(^{(6,13,21)}\). Mean HEI was significantly higher in the 42% of participants who consumed dietary supplements\(^{(13)}\). In one study, the HEI was used among various other indices (AHEI, DQI-R, RFS and aMED) to measure incidence of breast cancer\(^{(21)}\). However, it was shown to have limited ability to predict risk for oestrogen receptor (ER)-negative breast cancer\(^{(21)}\).

An Alternative HEI (AHEI) was used in two studies, with varied associations with cancer incidence\(^{(21,22)}\). It did not predict all-site cancer risk in a study of US adults\(^{(22)}\) but was inversely associated with chronic disease risk in men and women (39% and 28% reduced CVD risk in the highest AHEI quintile for men and women, respectively, with a weaker relationship in women). AHEI was associated with lower risk of ER-negative breast cancer in a study of postmenopausal women, with an 11% reduction in risk for each 10% increase in score\(^{(21)}\).

HEI derived from an FFQ (HEI-f) was used in two studies\(^{(23,24)}\). HEI-f was associated with an 11% reduction in major chronic disease risk in men (28%) and women (14%), but there were no associations with reduced cancer risk\(^{(23,24)}\).

**Healthy Diet Indicator**

The HDI was used in three studies\(^{(7,20,29)}\). One study used cognitive function as the outcome measure and found in
### Table 5  Prospective cohort studies with mortality and morbidity as outcome, ordered by increasing length of follow-up

<table>
<thead>
<tr>
<th>Study and reference</th>
<th>Dietary method</th>
<th>Subjects</th>
<th>Main outcome</th>
<th>Key findings</th>
<th>Mortality results</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trichopoulos et al. (2003)</td>
<td>FFQ</td>
<td>22043 adults aged 20–86 years. EPIC, Greece</td>
<td>Mortality: all-cause, all-site cancer and CHD</td>
<td>After 3-7-year follow-up MDS associated with reduced total mortality; 2-point increase in score led to 25% reduction. MDS inversely associated with mortality due to CHD and cancer</td>
<td>Cox proportional hazard model. Relative hazard (95% CI) of death for 2-point increment in MDS All-cause mortality</td>
<td>Possible residual confounding by unevaluated factors Short follow-up period for mortality outcome</td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td>Age/sex adj: 0.79 (0.69, 0.91) All adj: 0.75 (0.64, 0.87)</td>
<td></td>
</tr>
<tr>
<td>Trichopoulos et al. (1995)</td>
<td>FFQ</td>
<td>182 older adults aged &gt;70 years. Greece</td>
<td>All-cause mortality: survival beyond 70 years of age</td>
<td>After 4-year follow-up MDS inversely associated with all-cause mortality. For every 1-unit increase in score (maximum 8 points) all-cause mortality was significantly reduced by 17%</td>
<td>Cox proportional hazard model adjusted for age, sex, smoking and diet score For each unit increase in diet score, death rate ratio (95% CI) = 0.83 (0.69, 0.89) For men, relative to women, death rate ratio (95% CI) = 1.06 (0.55, 2.03)</td>
<td>Small cohort Short follow-up period for mortality outcome Study duration may not have been long enough to capture latency period needed to detect diet and cancer risk associations</td>
</tr>
<tr>
<td>Seymour et al. (2003)</td>
<td>FFQ</td>
<td>63,109 women and 52,724 men aged 50–79 years. American Cancer Society Cancer Prevention Study II Nutrition Cohort. USA</td>
<td>Mortality: short-term all-cause, circulatory and cancer</td>
<td>After 4-year follow-up DQI positively associated with all-cause and circulatory disease mortality in men and women, and cancer mortality in men only; when adjusted for multiple covariates relationship positive only for circulatory disease mortality in women. DQI unrelated to cancer mortality</td>
<td>Cox proportional hazard model. Death rate ratio (95% CI) for low diet quality v. high All-cause mortality</td>
<td>Limitations of DQI Short follow-up period for mortality outcome</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Age adj: 1.86 (1.28, 2.70) All adj: 1.23 (0.84, 1.81) All circulatory disease Age adj: 2.26 (1.12, 4.54) All adj: 1.81 (0.88, 3.72) All cancer Age adj: 0.93 (0.49, 1.77) All adj: 0.61 (0.32, 1.18)</td>
<td></td>
</tr>
<tr>
<td>Kant et al. (2000)</td>
<td>FFQ</td>
<td>42,254 women, mean age 61 years. Breast Cancer Detection Demonstration Project. USA</td>
<td>All-cause mortality</td>
<td>After 5-6-year follow-up RFS inversely related to all-cause and cause-specific mortality. 30% lower all-cause and cause-specific mortality in highest RFS quartile (age- and multivariate-adjusted)</td>
<td>Cox proportional hazard model. Relative risk (95% CI) for low (Q1) diet quality v. high (Q4) after exclusion for baseline disease All-cause mortality Age adj: 0.55 (0.45, 0.66) Multivariate adj: 0.66 (0.56, 0.82)</td>
<td>Generalising of results limited as cohort participants were from a screening study</td>
</tr>
<tr>
<td>Osler and Schroll (1997)</td>
<td>3d food record and frequency checklist</td>
<td>202 adults, mean age &gt;70 years. Denmark</td>
<td>All-cause mortality</td>
<td>After 6-year follow-up MDS inversely associated with all-cause mortality. A 1-unit increase in 7-point score reduced mortality by 21%. Plasma carotene levels significantly higher in subjects with high diet scores; plasma carotene negatively associated with mortality</td>
<td>Cox proportional hazard model For each unit increase in diet score, death rate ratio (95% CI)=0.79 (0.64, 0.98) For women, relative to men, death rate ratio (95% CI)=0.98 (0.54, 1.80)</td>
<td>Small cohort Study duration may not have been long enough to capture latency period needed to detect diet and cancer risk associations</td>
</tr>
<tr>
<td>Study and reference</td>
<td>Index</td>
<td>Dietary method</td>
<td>Subjects</td>
<td>Main outcome</td>
<td>Key findings</td>
<td>Mortality results</td>
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<tr>
<td>McCullough et al. (2000)(^{23})</td>
<td>HEI-f</td>
<td>131-item FFQ</td>
<td>51,529 men aged 40–75 years. Health Professionals’ Follow-Up Study. USA</td>
<td>Major chronic disease: incidence and mortality. Primary endpoint defined as non-fatal or fatal CVD, cancer or death (non trauma-related)</td>
<td>After 8-year follow-up HEI-f had weak inverse association with risk of major chronic disease (multiple covariate adjustment). 28% lower risk of CVD between highest and lowest HEI-f quintiles. 11% reduction in risk of developing CVD. No association with reduced cancer risk</td>
<td>Cox proportional hazard model. Relative risk (95% CI) for low (Q1) diet quality v. high (Q5)</td>
</tr>
<tr>
<td>McCullough et al. (2002)(^{22})</td>
<td>RFS</td>
<td>130-item FFQ</td>
<td>67,271 women and 38,615 men. Health Professionals’ Follow-Up Study and Nurses’ Health Study. USA</td>
<td>Major chronic disease defined as initial occurrence of cancer, CVD or non-traumatic death</td>
<td>After 8–12-year follow-up RFS and AHEI inversely related with chronic disease risk in men (covariate-adjusted). Men in highest AHEI quintile had 39% reduced risk of CVD v. lowest, while men in highest RFS quintile had 23% lower CVD risk (multivariate-adjusted). AHEI showed weak but significant inverse relationship with chronic disease risk in women; 28% lower CVD risk comparing highest AHEI quintile with lowest. RFS not related to major chronic disease in women. RFS and AHEI did not predict cancer risk in either sex</td>
<td>Relative risk for high (Q5) v. low diet quality (Q1) calculated with adjustment for other risk factors using pooled logistic regression</td>
</tr>
</tbody>
</table>

CVD
- AHEI – men
  - Age adj: 0.70 (0.63, 0.79)
  - All adj: 0.80 (0.71, 0.91)
- AHEI – women
  - Age adj: 0.78 (0.72, 0.84)
  - All adj: 0.89 (0.82, 0.96)
- RFS – men
  - Age adj: 0.79 (0.71, 0.88)
  - All adj: 0.93 (0.83, 1.04)
- RFS – women
  - Age adj: 0.84 (0.78, 0.91)
  - All adj: 0.98 (0.90, 1.06)

Cancer
- AHEI – men
  - Age adj: 0.52 (0.43, 0.63)
  - All adj: 0.61 (0.49, 0.75)
- AHEI – women
  - Age adj: 0.52 (0.44, 0.61)
  - All adj: 0.72 (0.60, 0.86)
- RFS – men
  - Age adj: 0.69 (0.58, 0.83)
  - All adj: 0.77 (0.64, 0.93)
- RFS – women
  - Age adj: 0.68 (0.58, 0.80)
  - All adj: 0.90 (0.75, 1.08)
<table>
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<th>Study and reference</th>
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<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fitzgerald et al. (2002)(^{11})</td>
<td>DQS (1)</td>
<td>24 h recall</td>
<td>2108 adults aged 18–74 years. Canada</td>
<td>Cancer incidence, all sites</td>
<td>After 8.3-year follow-up DQS had no relationship with incidence of all-sites cancer</td>
<td>Logistic regression to estimate OR of cancer for low (Q1) v. high (Q4) diet quality</td>
<td>Study duration may not have been long enough to capture latency period needed to detect diet and cancer risk associations</td>
</tr>
<tr>
<td>Mai et al. (2005)(^{32})</td>
<td>RFS</td>
<td>62-item FFQ</td>
<td>42254 women, mean age 61 years. USA</td>
<td>Cancer mortality and incidence: breast, lung, colorectal, other cancers</td>
<td>After 9.5-year follow-up RFS inversely associated with total mortality, cancer mortality, and mortality from colorectal, lung and breast cancer. Incidence of lung cancer reduced in highest RFS quartile</td>
<td>Cox proportional hazard model. Relative risk (95 % CI) for low (Q1) v. high (Q4) diet quality</td>
<td>Possible confounders: breast screening habits, treatment choice and lifestyle factors</td>
</tr>
<tr>
<td>Michels and Wolk (2002)(^{33})</td>
<td>RFS NRFS</td>
<td>60-item FFQ</td>
<td>59038 women aged 40–76 years. Mammography Screening Cohort, Sweden</td>
<td>Mortality: all-cause, CHD, stroke and cancer</td>
<td>After 9.9-year follow-up RFS inversely associated with all-cause and cause-specific mortality, particularly CVD and stroke. 42 % lower all-cause mortality in highest RFS quintile (adjusted for multiple covariates). Strong association between longevity and diet quality. NRFS not related to all-cause mortality, CVD and stroke mortality</td>
<td>Cox proportional hazard model. Hazard ratio (95 % CI) of death for highest diet quality (Q5) v. lowest (Q1)</td>
<td>Quantity and frequency of food consumption not considered</td>
</tr>
</tbody>
</table>

\(^{11}\) AHEI – women Age adj: 0.91 (0.83, 0.99) 
All adj: 0.97 (0.88, 1.06) 

\(^{32}\) RFS – men Age adj: 0.95 (0.82, 1.10) 
All adj: 1.08 (0.94, 1.25) 
RFS – women Age adj: 0.92 (0.85, 1.01) 
All adj: 1.00 (0.92, 1.11) 

\(^{33}\) Fitzgerald et al. \((2002)\) (11) DQS (1) 24 h recall 2108 adults aged 18–74 years. Canada 
Cancer incidence, all sites 
After 8.3-year follow-up DQS had no relationship with incidence of all-sites cancer 

\(^{34}\) Mai et al. \((2005)\) (32) RFS 62-item FFQ 42 254 women, mean age 61 years. USA 
Cancer mortality and incidence: breast, lung, colorectal, other cancers 
After 9.5-year follow-up RFS inversely associated with total mortality, cancer mortality, and mortality from colorectal, lung and breast cancer. Incidence of lung cancer reduced in highest RFS quartile 

\(^{35}\) Michels and Wolk \((2002)\) (33) RFS NRFS 60-item FFQ 59 038 women aged 40–76 years. Mammography Screening Cohort, Sweden 
Mortality: all-cause, CHD, stroke and cancer 
After 9.9-year follow-up RFS inversely associated with all-cause and cause-specific mortality, particularly CVD and stroke. 42 % lower all-cause mortality in highest RFS quintile (adjusted for multiple covariates). Strong association between longevity and diet quality. NRFS not related to all-cause mortality, CVD and stroke mortality 

\(^{36}\) Cox proportional hazard model. Relative risk (95 % CI) for low (Q1) v. high (Q4) diet quality | All cancer Men Age adj: 0.83 (0.44, 1.55) 
All adj: 0.81 (0.40, 1.64) 
Women Age adj: 0.74 (0.40, 1.38) 
All adj: 0.94 (0.44, 2.00) 

\(^{37}\) Cox proportional hazard model. Relative risk (95 % CI) for low (Q1) v. high (Q4) diet quality | All-cause mortality Crude: 0.67 (0.58, 0.77) 
Adj: 0.74 (0.63, 0.86) 
CVD mortality Crude: 0.58 (0.44, 0.75) 
Adj: 0.75 (0.57, 1.00) 
Stroke mortality Crude: 0.60 (0.42, 0.85) 
Adj: 0.71 (0.49, 1.03) 

\(^{38}\) Cox proportional hazard model. Relative risk (95 % CI) for low (Q1) v. high (Q4) diet quality | All cancer Men Age adj: 0.83 (0.44, 1.55) 
All adj: 0.81 (0.40, 1.64) 
Women Age adj: 0.74 (0.40, 1.38) 
All adj: 0.94 (0.44, 2.00) 

\(^{39}\) Cox proportional hazard model. Relative risk (95 % CI) for low (Q1) v. high (Q4) diet quality | All-cause mortality Crude: 0.66 (0.61, 0.73) 
Adj: 0.80 (0.73, 0.88) 
Cancer mortality Crude: 0.67 (0.58, 0.77) 
Adj: 0.74 (0.63, 0.86) 
CVD mortality Crude: 0.58 (0.44, 0.75) 
Adj: 0.75 (0.57, 1.00) 
Stroke mortality Crude: 0.60 (0.42, 0.85) 
Adj: 0.71 (0.49, 1.03)
<table>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Murphy et al. (1996)</strong>&lt;sup&gt;(34)&lt;/sup&gt;</td>
<td>DQ</td>
<td>24 h recall</td>
<td>6249 adults aged 45–74 years. NHANES I Epidemiologic Follow-up Study. USA</td>
<td>Survival beyond 45 years of age</td>
<td>After 10-year follow-up DQ inversely associated with all-cause mortality in males and females. Attenuated by employment, health, smoking and sociodemographic variables</td>
<td>Cox proportional hazard model adjusted for age. Relative hazard (95% CI) of death for poor diet quality v. all others</td>
<td>Use of 24 h recall as dietary measure</td>
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<td>Men 45–54 years: 1·5 (1·0, 2·3)</td>
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<td>55–64 years: 1·9 (1·4, 2·7)</td>
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<td>65–74 years: 1·4 (1·2, 1·6)</td>
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<td>Women 45–54 years: 1·5 (1·0, 2·4)</td>
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<td>55–64 years: 1·3 (1·0, 1·8)</td>
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<td>65–74 years: 1·2 (1·0, 1·4)</td>
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</tr>
<tr>
<td><strong>Osler et al. (2002)</strong>&lt;sup&gt;(30)&lt;/sup&gt;</td>
<td>HFI</td>
<td>26-item FFQ</td>
<td>7316 adults aged 30–70 years. Denmark</td>
<td>Fatal and non-fatal CHD</td>
<td>Follow-up varied from 4 to 14 years. HFI had an insignificant, inverse association with CVD risk</td>
<td>Cox proportional hazard model. Hazard rate ratio (95% CI) for CVD for high (Q4) v. low (Q1) diet quality Crude: 0·70 (0·47, 1·03) Adj: 1·21 (0·80, 1·82)</td>
<td>Possible dietary measurement bias of short FFQ</td>
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<td><strong>Bazelmans et al. (2006)</strong>&lt;sup&gt;(31)&lt;/sup&gt;</td>
<td>HFNI</td>
<td>1 d food record</td>
<td>Adult men and women. Belgium</td>
<td>Mortality: all-cause</td>
<td>After 10-year follow-up HFNI significantly associated with all-cause mortality in men (adjusted for risk factors). Risk of death decreased from quartile 1 to 4. HFNI not significant in women</td>
<td>OR for total mortality derived from logistic regression models for low (Q1) v. high (Q4) diet quality Men Adj age: 1·53 (1·10, 2·12) Adj all: 1·68 (1·19, 2·37) Women Adj age: 1·03 (0·57, 1·82) Adj all: 1·05 (0·58, 1·87)</td>
<td>Use of 1 d food record as dietary measure Scoring may not best represent data Lack of definition in some index components Dietary guidelines used for basis of HFNI may not adequately describe intake associated with reduced risk of mortality</td>
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<tr>
<td>Study and reference</td>
<td>Index</td>
<td>Dietary method</td>
<td>Subjects</td>
<td>Main outcome</td>
<td>Key findings</td>
<td>Mortality results</td>
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| Knoops et al. (2006)
(20) | MDS (4) MAI HDI | Diet history | Elderly: 2068 men and 1049 women aged 70–90 years. HALE study | Mortality: all-cause, CVD and other causes | After 10-year follow-up MDS, MAI and HDI significantly inversely related to all-cause mortality. Northern Europe had higher absolute mortality risk compared with Southern Europe. MAI had stronger association with all-cause mortality in Northern Europe compared with Southern Europe | Cox proportional hazard model. Hazard rate ratio (95 % CI) for high (âŠ¥median) diet quality v. low (<median) | Study duration may not have been long enough to capture latency period needed to detect diet and cancer risk associations |
| McCullough et al. (2000)
(24) | HEI-f | 116-item FFQ | 62,722 women aged 30–55 years. Nurses’ Health Study. USA | Major chronic disease: defined as non-fatal or fatal CVD, cancer or death (non trauma-related) | After 12-year follow-up HEI-f was not associated with major chronic disease risk (covariate-adjusted). HEI-f highest quintile was associated with a 14 % lower risk for CVD, with no association with reduced cancer risk | Cox proportional hazard model. Relative risk (95 % CI) for low (Q1) diet quality v. high (Q5) Major chronic disease | Study duration may not have been long enough to capture latency period needed to detect diet and cancer risk associations |
| Lagiou et al. (2006)
(18) | MDS (2) | 80-item FFQ | 42,237 young women aged 30–49 years. Swedish section of Scandinavian Women’s Lifestyle and Health Cohort. Sweden | Mortality: all-cause and cancer | After 12-year follow-up, no association between women aged 30–49 years at enrolment and overall or cancer mortality. 2-point increase in MDS inversely associated with substantial reduction in all-cause mortality (23 %) and cancer mortality (29 %) in women aged 40–49 years at enrolment | Cox proportional hazard model. Hazard rate ratio (95 % CI) for bottom third of diet quality v. top Total mortality Cancer mortality | Possible dietary changes during the follow-up period |

Lack of definition in some index components
Mortality associations assessed in relation to dichotomous ranking of high and low quality at the median score
Study duration may not have been long enough to capture latency period needed to detect diet and cancer risk associations
Some HEI-f components weaken associations with chronic disease outcomes
Lack of diversity in cohort population
Possible dietary changes during the follow-up period
Lack of definition in some index components
Type of alcohol not defined; protective benefits cannot be assumed
Table 5 Continued

<table>
<thead>
<tr>
<th>Study and index</th>
<th>Dietary index method</th>
<th>Subjects</th>
<th>Main outcome</th>
<th>Key findings</th>
<th>Mortality results</th>
<th>Limitations</th>
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<tbody>
<tr>
<td>Huijbregts et al. (1997)</td>
<td>HEI (1989)</td>
<td>3045 men aged 50–70 years</td>
<td>All-cause mortality</td>
<td>After 20-year follow-up HDI inversely associated with all-cause and cardiovascular mortality. Multivariate-adjusted risk of all-cause mortality reduced by 13% in highest HDI group relative to lowest. HDI overall was associated with lower mortality.</td>
<td>Relative risk (95% CI) of all-cause mortality: All adj: 0.88 (0.79, 0.97)</td>
<td>Potential influence of HDI on mortality and morbidity is not clear. Limited external validity due to cohort participants and study design.</td>
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<tr>
<td>Fung et al. (2001)</td>
<td>AHEI</td>
<td>71,058 women, Nurses’ Health Study, USA</td>
<td>Mortality: CVD, breast cancer</td>
<td>After 18-year follow-up, AHEI associated with lower risk of ER-positive breast cancer for low (Q1) diet quality relative to higher (Q5) diet quality. Adjusted relative risk (95% CI) for ER-positive breast cancer for low (Q1) diet quality relative to higher (Q5) diet quality: Adjusted: 0.82 (0.62, 1.07).</td>
<td>Relative risk (95% CI) of all-cause mortality for low (Q1) diet quality relative to high (Q5) diet quality: Adjusted: 0.88 (0.79, 0.97).</td>
<td>Potential influence of AHEI on mortality and morbidity is not clear. Limited external validity due to cohort participants and study design.</td>
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<td>Odel et al. (2001)</td>
<td>DGI</td>
<td>7316 adults aged 30–70 years</td>
<td>Mortality: all-cause mortality</td>
<td>After 13-year follow-up, DGI inversely associated with all-cause mortality. Cox proportional hazard model. Adjusted relative risk (95% CI) for all-cause mortality for low (Q1) diet quality relative to high (Q5) diet quality: Crude: 0.60 (0.43, 0.86).</td>
<td>Cox proportional hazard model. Adjusted relative risk (95% CI) for all-cause mortality for low (Q1) diet quality relative to high (Q5) diet quality: Crude: 0.60 (0.43, 0.86).</td>
<td>Potential influence of DGI on mortality and morbidity is not clear. Limited external validity due to cohort participants and study design.</td>
</tr>
<tr>
<td>Harwalkar et al. (2002)</td>
<td>FFQ</td>
<td>37,084 postmenopausal women, Iowa, USA</td>
<td>Mortality: all-cause mortality</td>
<td>After 13-year follow-up, FFQ inversely associated with all-cause mortality. Cox proportional hazard model. Adjusted relative risk (95% CI) for all-cause mortality for low (Q1) diet quality relative to high (Q5) diet quality: Crude: 0.60 (0.43, 0.86).</td>
<td>Cox proportional hazard model. Adjusted relative risk (95% CI) for all-cause mortality for low (Q1) diet quality relative to high (Q5) diet quality: Crude: 0.60 (0.43, 0.86).</td>
<td>Potential influence of FFQ on mortality and morbidity is not clear. Limited external validity due to cohort participants and study design.</td>
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four of five cohorts that a higher HDI was associated with lower cognitive impairment. However, this was statistically significant in only one cohort \(^{29}\). Two studies found the HDI to be inversely associated with all-cause mortality \(^{27,20}\). One found a 13% reduction in all-cause mortality as well as an 18% risk reduction in CVD mortality, with no reduction in cancer mortality risk \(^{7}\). The other found that the HDI had a significant inverse relationship with all-cause mortality after a 10-year follow-up \(^{20}\).

**Healthy Food Index**

The HFI was used in two assessments of the same population, but reported associations with different outcomes \(^{8,30}\). The first measured CVD mortality \(^{8}\) while the second measured risk of CHD \(^{30}\). HFI was inversely associated with all-cause mortality in men and women; however, this was attenuated after adjustment for smoking, BMI, physical activity, alcohol intake and education level \(^{8}\). It was not related to cause-specific mortality \(^{8}\). The second report found an insignificant, inverse relationship with CHD risk \(^{30}\).

The Healthy Food and Nutrient Index (HFNI) was used in one study \(^{31}\). It had a significant association with mortality in men after adjustment for all-cause mortality risk factors. This index showed no significant associations in women.

**Recommended Food Score**

The original RFS was used in three studies \(^{49,22,32}\). RFS was inversely associated with all-cause mortality and cause-specific mortality in a study of US women \(^{9}\). There was a 30% reduced risk of all-cause and cause-specific mortality in the highest quartile, after age and multivariate adjustment \(^{9}\). One of the studies that measured AHEI also found that the RFS was inversely associated with chronic disease risk in men, with a 23% lower CVD risk, and that this was a smaller association than with AHEI \(^{22}\). The RFS was not related to major chronic disease outcomes in women and did not predict cancer in either gender \(^{22}\). In contrast to the previous study, another in US women found the RFS to be inversely associated with total mortality, cancer mortality, and mortality from colorectal, lung and breast cancer, with the highest RFS quartile having a reduced incidence of lung cancer \(^{32}\).

Two studies using a slightly varied version of RFS also found associations with mortality and cancer \(^{21,33}\). It was inversely associated with all-cause and cause-specific mortality, particularly CHD and stroke, with 42% reduced all-cause mortality \(^{33}\). One study previously mentioned found the RFS to be associated with a lower risk of ER-negative breast cancer (12% reduction for each 10% increase in score), and that it had the strongest association among all the indices used (HEI, AHEI, DQI-R and aMED) \(^{21}\).

A Not Recommended Food Score (NRFS) was used in a previous study \(^{33}\) using the RFS, which showed no relationships with all-cause mortality, CHD and stroke mortality.
**Diet Quality Index**

The original DQI was used in one study of US adults. After adjustment for multiple covariates the DQI was associated only with circulatory disease mortality in women and it was unrelated to cancer mortality. An improved index, the DQI Revised (DQI-R), was limited in predicting risk of breast cancer. Another version of the DQI was modified for use in a Mediterranean population with dietary biomarkers as an outcome. It was found to be significantly correlated with EPA and DHA only. This association was still apparent after adjusting for tobacco use, and a correlation with β-carotene became significant after this adjustment.

**Diet Quality Score**

Two variations of the DQS have been used. The first measured all-sites cancer incidence over 8-3 years and found a significant inverse relationship, estimating that the incidence of cancer could be reduced by ∼35% if diet quality was improved. The second, using CVD biomarkers, found DQS to be negatively associated with total cholesterol, LDL cholesterol, TAG, homocysteine and absolute risk of IHD after adjustment for age, sex, physical activity level and smoking. DQS was positively associated with HDL cholesterol concentrations. Another score, the Dietary Quality (DQ), was used to examine associations with survival after age 45 years. The latter study in US adults found that DQ was inversely associated with all-cause mortality, with the relative hazards of death for men aged 55–64 years with the worst DQ after 10-year follow-up being 1.9 compared with those with better diets. The relative hazards of death for men aged 45–54 and 65–74 years were 1.5 and 1.4, respectively.

**Dietary Guidelines Index**

The DGI was used in one study to measure all-site cancer and site-specific cancer incidence and mortality. Higher DGI was associated with a significantly lower incidence of all-site cancer and site-specific cancers. This association was no longer significant once guidelines such as be physically active each day were excluded and it was analysed against only diet-based dietary guidelines and cancer incidence, with the exception of lung and bronchus.

**Mediterranean Diet Score**

Numerous versions of MDS have been constructed, most with slight variations from the original, mainly in the scoring of fish, legumes and vegetables, as detailed in Table 1. Six studies used the MDS with most measuring all-cause mortality and cancer mortality as outcomes. The first version found that every 1-unit increase in score was significantly associated with reduced total mortality (25% reduction for a 2-point increase in score) and inversely associated with mortality due to CHD and cancer. The second study in Southern Europe found an inverse relationship, with a substantial reduction in all-cause mortality (23%) and cancer mortality (29%) in women aged 40–49 years at enrolment. This association was not found in women who were aged 30–40 years at enrolment. A third version of the MDS was used to measure all-cause mortality as an outcome. It too was inversely associated with all-cause mortality; a 1-unit increase in score being associated with 21% reduced mortality. The study also found higher plasma carotene levels in subjects with higher scores and a negative association between plasma carotene and mortality. The fourth version of the MDS was used in a study that also used the HDI and Mediterranean Adequacy Index (MAI). All three indices had a significant inverse relationship with all-cause mortality. An Alternative MDS (aMDS) was used in a previous study on breast cancer and was associated with lower risk of ER-negative breast cancer (7% reduction for each 10% increase in score). One study used the MAI to measure all-cause mortality, cancer and other causes of mortality. Only all-cause mortality had a significant inverse relationship with MAI, with associations found to be stronger in Northern than in Southern Europe.

**Total and specific food group diversity**

Three case–control studies of colon and colorectal cancer used total and specific food group diversity. The first study from Italy found total diet diversity reduced the risk of colorectal cancer by 30% in the highest quartile of diversity. Relative risk appeared to increase by 1.4 with diversity of foods in the carbohydrate group. The second study found that total diet diversity had no association with colon cancer in US adults. However, men had a 50% increased risk of colon cancer with greater diet diversity in the meat/fish/poultry/egg group and refined grains group. Women had a 20% lower risk if diet diversity was high in the vegetable group. The third study, also in Italy, found total diet diversity to be inversely related to colon cancer risk in men (35% reduction) and that diversity within the vegetable group had an inverse relationship with both colon and rectal cancers.

**Discussion**

The majority of studies reviewed found that diet quality scores were inversely related to health outcomes, with a protective effect of moderate magnitude. In the studies that did find associations, all-cause mortality was reduced by 17–42%, CVD mortality by 18–53%, CVD risk by 14–28%, cancer mortality by 13–30%, and all-cancer risk.
by 7–35%. The predictive capacity of most indices appears to be in a similar range. However, it is difficult to directly compare results due to the variability of population groups, length of study follow-up periods, dietary measurement methods, index scoring methods and approaches to adjustment for confounders. Relative risks were consistently attenuated after adjustment for factors such as BMI, physical activity, age, education, socio-economic status and smoking.

Associations between scores and morbidity and/or mortality varied between genders within studies, and were generally stronger and broader in their associations with adverse health outcomes for men. This suggests that this is a real disparity, although differential gender bias in the original dietary assessment method used to assess usual intake is a possibility.

Specific outcomes, such as cancer risk, were not predicted as strongly or consistently as all-cause mortality or CVD risk. Those indices that did predict risk came from tools based on either a US dietary pattern (RFS) and AHEI, and Total Diet Diversity (MDS and aMED) or Mediterranean dietary patterns (MDS and aMED). One possible reason for the lack of association is study duration, with shorter follow-up obviously not able to capture the latency period prior to cancer detection. Two of the studies that did find associations with cancer risk were case–control studies that measured the diet diversity of participants after initial cancer diagnosis through administration of an FFQ covering the previous year. However, dietary intake in these instances is likely to be confounded by recall bias and it is difficult to ascertain with this study design in which direction the risk assessment is likely to be affected. All indices have limitations due to the methods with which dietary intake was measured. Instruments such as the 24 h recall and FFQ all have limitations including over- or under-reporting, and in some studies only a brief FFQ was originally used or sometimes a single 24 h recall was used as the indicator of usual dietary intake. Having an initial measurement of dietary intake at baseline in cohort studies is a more sensitive way to ascertain prospective associations with disease risk. However, this approach requires long periods of follow-up and is also not without bias and limitations. To strengthen comparisons across studies, researchers ideally could base their index of diet quality on one of the tools commonly used or use more than one of the indices in statistical analyses, as some researchers have done. The critical appraisal of study quality found that most studies met at least three of five criteria, with about a third meeting at least four. While outcomes were usually reported objectively and statistical methods appeared appropriate, descriptions of the population sampling, inclusion criteria and subgroups were often poor.

Many components of indices such as the HEI are derived from epidemiological associations with reduced risk of CVD and its risk factors, as opposed to cancer. This may explain why indices appear to have a better risk prediction for CVD. Associations may also be less strong between index components and cancer risk. Most indices have been developed to measure diet quality and adherence to national dietary guidelines (HFNI, RFS, DQI and DGI). While they attempt to show association with higher diet quality, it is difficult to predict the likely degree of association with disease risk if the index was not specifically designed for this purpose. For example in the HEI, grains are not differentiated between those that are refined or unrefined, which limits the ability to show effects between whole grains and diseases such as diabetes and heart disease.

Indices have their own strengths and limitations which may have affected results in the studies. Indices that have a small scoring scale, such as the DQI and HEI, appear to be less sensitive in this evaluation of indices and fail to capture extremes and intrinsic characteristics of food behaviours or eating patterns. Their discrete distributions within index scoring patterns may reduce their power as a predictor. Both the DQI and the HEI have been revised (DQI-R and AHEI) to improve disease risk prediction. AHEI was twice as strong as HEI in its overall chronic disease risk prediction, primarily CVD, in men and women in the USA. When selecting or developing a dietary quality index, important considerations include: (i) defining the tool’s purpose, such as to capture a global indicator of adherence to specific aspects of a healthful diet or to measure associations with aspects of diet that increase disease risk; (ii) identifying a scoring system that weights the subscales appropriately; and (iii) measuring potential confounders to allow appropriate adjustment for confounders. Further, consideration should be given to including a validation of the score using appropriate biomarkers and disease risk factor assessment. For example, a dietary quality index to measure associations with CVD outcomes could include index components that score type and quantity of dietary fat, measure red blood cell membrane fatty acids as the dietary biomarkers, plasma lipids as the disease risk factors and confounders of CVD as previously described.

Other limitations of study design include small cohort size, cross-sectional assessments only with no follow-up and using mortality as the sole outcome, given that fatality may be confounded by early diagnosis, treatment and medical care. Therefore indices need to be carefully interpreted to ensure limitations are acknowledged.

Conclusions

Indices of diet quality are used to measure associations with biomarkers and health outcomes. We found that lower diet quality scores are consistently associated with
higher rates of all-cause mortality and selected disease-specific rates or mortality. The associations are attenuated when adjusted for common confounding variables but still remain significant, and appear to be stronger in men and for all-cause and CVD mortality. However, the limitations of these indices and the specific context in which they are used need to be considered when interpreting results and comparing studies. Future validation studies need to examine associations between nutritional biomarkers and intermediary disease risk factors. Not only will this improve the validity of the diet quality indices, but it will also increase their potential practical applications in both clinical and public health contexts.

As there are numerous diet quality indices and variations of each method, it is recommended that researchers model indices on existing tools and select more than one when testing associations with health outcomes. This will allow examination of how robust findings are across the indices and facilitate comparison across studies.

Finally, there is enough evidence to recommend that diet quality tools should be adapted for use in clinical dietetic practice and for self-evaluation of dietary intake, particularly those scored in a way that identifies which foods need to be increased to obtain a more healthful score and therefore potentially reduce chronic disease risk.

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References