Food groups and risk of all-cause mortality: a systematic review and meta-analysis of prospective studies[1,2]

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ABSTRACT

Background: Suboptimal diet is one of the most important factors in preventing early death and disability worldwide.

Objective: The aim of this meta-analysis was to synthesize the knowledge about the relation between intake of 12 major food groups, including whole grains, refined grains, vegetables, fruits, nuts, legumes, eggs, dairy, fish, red meat, processed meat, and sugar-sweetened beverages, with risk of all-cause mortality.

Design: We conducted a systematic search in PubMed, Embase, and Google Scholar for prospective studies investigating the association between these 12 food groups and risk of all-cause mortality. Summary RRs and 95% CIs were estimated with the use of a random effects model for high-intake compared with low-intake categories, as well as for linear and nonlinear relations. Moreover, the risk reduction potential of foods was calculated by multiplying the RR by optimal intake values (serving category with the strongest association) for risk-reducing foods or risk-increasing foods, respectively.

Results: With increasing intake (for each daily serving) of whole grains (RR: 0.92; 95% CI: 0.89, 0.95), vegetables (RR: 0.96; 95% CI: 0.95, 0.98), fruits (RR: 0.94; 95% CI: 0.92, 0.97), nuts (RR: 0.76; 95% CI: 0.69, 0.84), and fish (RR: 0.93; 95% CI: 0.88, 0.98), the risk of all-cause mortality decreased; higher intake of red meat (RR: 1.10; 95% CI: 1.04, 1.18) and processed meat (RR: 1.23; 95% CI: 1.12, 1.36) was associated with an increased risk of all-cause mortality in a linear dose-response meta-analysis. A clear indication of nonlinearity was seen for the relations between vegetables, fruits, nuts, and dairy and all-cause mortality. Optimal consumption of risk-decreasing foods results in a 56% reduction of all-cause mortality, whereas consumption of risk-increasing foods is associated with a 2-fold increased risk of all-cause mortality.

Conclusion: Selecting specific optimal intakes of the investigated food groups can lead to a considerable change in the risk of premature death. *Am J Clin Nutr* 2017;105:1462–73.

Keywords: food groups, diet, meta-analysis, dose response, mortality

INTRODUCTION

In 2013, the number of deaths worldwide and among all age groups amounted to nearly 55 million; 70% of these were caused by noncommunicable diseases (1). One-third of these fatalities were caused by cardiovascular disease (CVD), followed by cancer at 15% (1). A high-quality diet comprising abundant amounts of whole grains, fruits, vegetables, nuts, and fish is one of the most important factors in preventing early death and disability worldwide (2).

During the past 50 y, lifestyle factors have been identified as modifiable factors associated with death. Thus, despite often unclear direct biological mechanisms due to the many potential underlying disease mechanisms, epidemiologic risk factors that can change the probability of death are important public health indicators. Studies that were able to translate risk reduction into measures of life expectancy calculated that populations with a low-risk profile (no smoking, physically active, healthy dietary pattern) differ in 10–15 y from those with a high-risk profile (3).

Previous meta-analyses showed that whole grains, fruits and vegetables, nuts, and fish were associated with a lower risk of all-cause mortality (4–7), whereas red and processed meats were associated with an increased risk (8). In addition, a recent meta-analysis showed that high adherence to diet quality indexes such as the Healthy Eating Index and Dietary Approaches to Stop Hypertension were associated with a 22% decrease in the risk of all-cause mortality (9).

When investigating diet-disease relations in terms of their meaning for public health, the best approach is the study of foods or food groups instead of nutrients. Moreover, foods are

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[2] Supplemental Material 1, Supplemental Tables 1–26, and Supplemental Figures 1–31 are available from the "Online Supporting Material" link in the online posting of the article and from the same link in the online table of contents at http://ajcn.nutrition.org.

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directly linked to food-based dietary guidelines, which are based on the preventive actions of foods and should be released for each country depending on dietary practices and intake amounts (10). Evidence for food consumption–death risk relations from systematic reviews is a key component of this process.

Up-to-date evidence about the association of all-cause mortality risk with consumption of legumes, eggs, and sugar-sweetened beverages (SSBs) has not been synthesized. Moreover, meta-analyses rarely assess the quality of evidence. Because meta-analyses are a crucial building block in generating guidelines and recommendations, it is of the utmost importance to implement a method to evaluate the quality of evidence.

Thus, we investigated the associations of 12 food groups defined a priori, including whole grains, refined grains, vegetables, fruits, nuts, legumes, eggs, dairy, fish, red meat, processed meat, and SSBs, with risk of all-cause mortality by evaluating all available data from prospective studies. Special attention was given to the strength and shape of the dose-response relationship for finding an optimal intake for lowest all-cause mortality risk. Finally, using the NutriGrade scoring system, we aimed to determine the quality of meta-evidence of the protective or detrimental effects of the food groups on all-cause mortality risk.

METHODS

The review was registered in PROSPERO International Prospective Register of Systematic Reviews (www.crd.york.ac.uk/PROSPERO/; identifier CRD42016037069). This systematic review was planned and conducted according to the standards of the Meta-analysis of Observational Studies in Epidemiology (11).

Search strategy

The literature published through December 2016 was searched using the electronic databases PubMed, Embase, and Google Scholar, with no restrictions on language or calendar date. The search terms used are listed in **Supplemental Material 1**.

Moreover, the reference lists from the retrieved articles, systematic reviews, and meta-analyses were checked for further relevant studies. The literature search was conducted by 2 authors (LS and A-ML); disagreements were resolved by consensus after discussion with another reviewer (HB).

Study selection

Studies were included in the meta-analysis if they *1*) were cohort studies, case-cohort studies, follow-ups of randomized controlled trials, and case-control studies nested in a prospective study that were peer-reviewed and for which the full text was available; *2*) provided information about the association for ≥1 of the following 12 food groups: whole grains and cereals, refined grains and cereals, vegetables, fruits, nuts, legumes, eggs, dairy products, fish, red meat, processed meat, and SSBs [these 12 food groups are the focus because most diet quality indexes or scores are based on them (9, 12, 13), as previously reported (14)]; *3*) included participants aged ≥18 y; and *4*) considered all-cause mortality as an outcome. We excluded studies including populations suffering from chronic disease and studies reporting only cause-specific mortality.

Data extraction

After selecting studies, 2 reviewers (LS and A-ML) extracted the following information: name of first author, year of publication, study origin, cohort name, sample size, number of subjects, age at entry, sex, study duration (follow-up in years), outcome, outcome assessment, assessment of food group, quantity of food, risk estimate [most adjusted measures; HRs, RRs, or ORs with their corresponding 95% CIs], and adjustment factors.

When a study provided several risk estimates, the multivariable adjusted model was chosen. When only separate risk estimates for male and female participants were available in a study, we combined the RRs using a fixed effects model before inclusion in the meta-analysis.

Risk of bias assessment

To determine the risk of bias of the prospective studies, we assessed how the studies ascertained exposure, how they assessed outcomes, whether the follow-up was adequate (≥10 y), and whether they adjusted the basic model and made any outcome-relevant adjustments (age, sex, education, BMI, smoking, physical activity, or energy intake) (15). Studies were classified as being at low risk of bias in general only if none of the domains established a high or unclear risk of bias.

Statistical analysis

A random effects model was used to calculate summary RRs and 95% CIs for the associations between all-cause mortality and the highest compared with the lowest intake category for each of the 12 food groups (whole grains and cereals, refined grains and cereals, vegetables, fruits, nuts, legumes, eggs, dairy products, fish, red meat, processed meat, and SSBs) and for the dose-response analysis (16), which incorporated both within-study and between-study variabilities. To evaluate the weighting of each study, the SE for the logarithm RR for each study was calculated and regarded as the estimated variance of the logarithm RR, through the use of an inverse variance method (16).

A method described by Greenland and Longnecker (17) and Orsini et al. (18) was applied for the dose-response analysis; this computed study-specific slopes (linear trends) and 95% CIs from the natural logs of the RRs and CIs across intake categories for the 12 food groups. The method requires knowing the distribution of cases and person-years or noncases and the RRs with the 95% CIs for ≥3 quantitative exposure categories.

When studies reported only the total number of cases or total person-years and the exposure was defined in quantiles, the distribution of cases or person-years was calculated by dividing the total number by the number of quantiles. Whenever reported, the mean or median intake by category was assigned to the corresponding RR. The midpoint was calculated for studies that reported only a range of intake by category. When the intake values were open-ended, we assumed that their range was the same as that of the adjacent category. For studies presenting the exposure per given unit of energy intake, we rescaled the exposure using the mean energy intake provided.

The dose response was expressed in the following servings: 30 g whole grains or cereals/d, 30 g refined grains or cereals/d, 100 g vegetables/d, 100 g fruits/d, 28 g nuts/d, 50 g legumes/d, 50 g eggs/d, 200 g dairy products/d, 100 g fish/d, 100 g red meat/d,

50 g processed meat/d, and 250 mL SSBs/d. For studies that reported intake only as serving size (and did not specify the quantitative amount), we used recommended conversions (**Supplemental Table 1**).

To examine possible nonlinear associations, we calculated restricted cubic splines for each study with ≥3 categories of exposure, using 3 fixed knots at 10%, 50%, and 90% through the total distribution of the reported intake, and combined them using multivariate meta-analysis (19).

Moreover, the risk reduction potential of foods was calculated by multiplying the RR by optimal intake values (serving category with the strongest association) of risk-decreasing foods ($RR^{*}_{reduced}$), and risk-increasing foods ($RR^{*}_{increased}$), respectively. The optimal intake value was defined as the number of servings of a single food group with the strongest association for all-cause mortality.

To explore heterogeneity between studies, we used the Cochran Q test and the I^2 statistic [with I^2 >50% considered to represent potentially important statistical heterogeneity (20)]. In addition, to identify potential sources of heterogeneity, we stratified the meta-analysis by subgroups: sex, duration of follow-up (mean or median ≥10 compared with <10 y), geographic location (Europe, America, Asia, or Australia), number of cases (≥1000 compared with <1000), and dietary assessment (validated compared with nonvalidated). Furthermore, we analyzed sensitivity for studies with a low risk of bias.

Potential small-study effects such as publication bias were explored using the Egger test and funnel plots (21) if ≥10 studies were available, as recommended by the Cochrane Handbook (22).

Review Manager 5.3 (Nordic Cochrane Center), and Stata software version 14 (StataCorp) were used for the statistical analyses.

Quality of meta-evidence

To evaluate the meta-evidence for the association between 12 predefined food groups and all-cause mortality (quality of evidence of meta-analyses was defined as the confidence in the estimate), we applied the NutriGrade scoring system (a maximum of 10 points), which comprises the following items: *1*) risk of bias, study quality, and study limitations; *2*) precision; *3*) heterogeneity; *4*) directness; *5*) publication bias; *6*) funding bias; *7*) study design (only for meta-analyses of randomized controlled trials); and *8*) effect size (15). Based on this scoring system, we recommend 4 categories to judge the meta-evidence: high (≥8 points), moderate (6 to <8 points), low (4 to <6 points), and very low (0 to <4 points).

RESULTS

Of the 17,579 records that were identified by the literature search, 266 full-text articles were assessed in detail because they reported ≥1 of the 12 food groups and all-cause mortality in the title or abstract (**Figure 1**).

Included in the meta-analysis were 19 prospective studies (18 reports) of consumption of whole grains (23–40) (**Supplemental Table 2**), 4 studies of refined grains (27, 30, 33, 36) (**Supplemental Table 3**), 37 studies of vegetables (36 reports) (23–25, 29, 31, 34–36, 38, 39, 41–66) (**Supplemental Table 4**), 34 studies of fruits (33 reports) (23–25, 29, 31, 34–36, 38, 39, 41, 42, 45, 46, 48, 49, 51–67) (**Supplemental Table 5**), 16 for nuts (14 reports) (36, 42, 68–79) (**Supplemental Table 6**), 17 studies of legumes (16 reports) (34, 36, 42, 47, 50, 51, 56, 57, 63, 65, 78, 80–84) (**Supplemental Table 7**), 8 studies of egg consumption (36, 56, 77, 78, 80, 85–87) (**Supplemental Table 8**), 27 studies of dairy products (25 reports) (23, 24, 35, 44, 46–48, 50, 65, 77, 78, 80, 82, 84, 88–98) (**Supplemental Table 9**), 39 studies of fish (37 reports) (31, 34, 35, 38, 39, 42, 46, 50, 53, 56, 59, 61, 63–65, 77, 80, 81, 99–117) (**Supplemental Table 10**), 12 studies of red meat (10 reports) (42, 61, 105, 106, 112, 118–122) (**Supplemental Table 11**), 7 studies of processed meat (6 reports) (61, 105, 119–122) (**Supplemental Table 12**), and 5 studies of SSBs (78, 95, 123–125) (**Supplemental Table 13**).

Whole grains

Nineteen studies with 121,141 mortality cases were included in the high- compared with low-intake meta-analysis (overall intake range: 0−110 g/d). When we compared extreme categories, an inverse association between all-cause mortality and whole-grain intake was observed, with an indication for high heterogeneity between studies (RR: 0.88; 95% CI: 0.84, 0.92; I^2 = 91%; *P*-heterogeneity < 0.001) (**Supplemental Figure 1**). Each additional daily 30 g of whole grains was inversely associated with mortality risk (RR: 0.92; 95% CI: 0.89, 0.95; I^2 = 80%; *P*-heterogeneity < 0.001; *n* = 11) (**Supplemental Figure 2**). The heterogeneity persisted in additional analyses stratified by sex, follow-up duration, geographic location, number of cases, and dietary assessment (**Supplemental Table 14**). The inverse association was not observed in European, Asian, Australian, or short-term studies. These subgroup differences were not statistically significant. Some evidence of heterogeneity between studies applying validated compared with nonvalidated dietary assessment methods was observed. An inverse association was observed only for studies applying validated dietary assessment methods.

There was significant evidence for small study effects in the high-intake compared with the low-intake meta-analysis (*P* = 0.02) but not in the dose-response meta-analysis (*P* = 0.64). Visual inspection of the funnel plot (dose-response analysis) indicated moderate symmetry (**Supplemental Figure 3**). Although the test for nonlinearity was significant (*P*-nonlinearity < 0.01; *n* = 10 studies), a clear dose-response relation was observed. The risk of mortality decreased by 25% with increasing intake of whole grains up to ∼100 g/d (**Figure 2**).

Refined grains

Four studies with 11,034 mortality cases were included in the high-intake compared with low-intake meta-analysis (overall intake range: 0−183 g/d). No association was observed for the highest compared with the lowest refined grain intake category (RR: 0.99; 95% CI: 0.94, 1.05; I^2 = 26%; *P*-heterogeneity = 0.26) (**Supplemental Figure 4**), or for each additional daily 30 g (RR: 0.99; 95% CI: 0.97, 1.01; I^2 = 7%; *P*-heterogeneity = 0.36; *n* = 4) (**Supplemental Figure 5**). No significant association was observed in any of the stratified analyses (**Supplemental Table 15**). Furthermore, no evidence of a nonlinear dose-response association was found (*P*-nonlinearity = 0.11; *n* = 3 studies) (Figure 2).

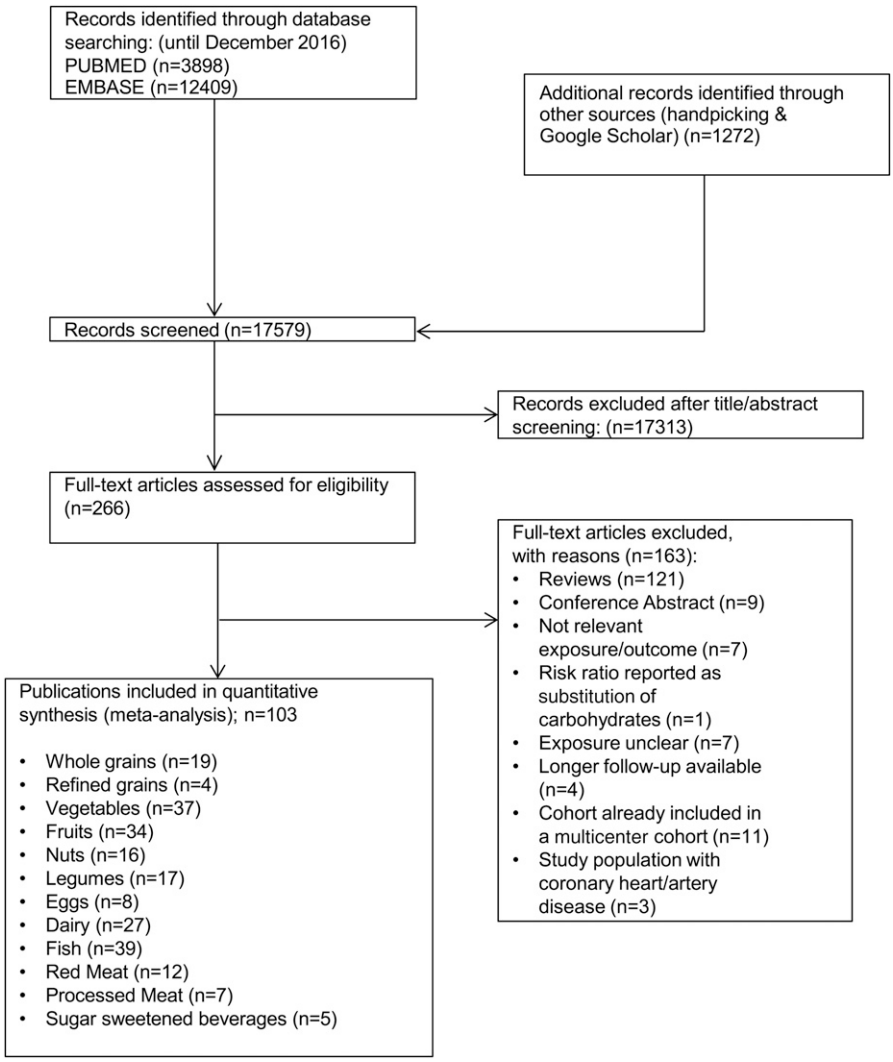

FIGURE 1 Flowchart of study selection.

Vegetables

Thirty-seven studies with 121,067 mortality cases were included in the high-intake compared with the low-intake meta-analysis (overall intake range: 5−663 g/d). An inverse association was observed for the high intake compared with the low intake (RR: 0.93; 95% CI: 0.90, 0.95; I^2 = 75%; P-heterogeneity < 0.001) (**Supplemental Figure 6**) and dose-response analyses (RR/100 g: 0.96; 95% CI: 0.95, 0.98; I^2 = 67%; P-heterogeneity < 0.001; n = 17) (**Supplemental Figure 7**).

The observed heterogeneity persisted largely in additional analyses stratified by sex, follow-up duration, geographic location, and number of cases. The inverse association was not observed in studies conducted in the United States or in studies including only women (**Supplemental Table 16**). No evidence of heterogeneity was detected between subgroups in stratified analyses.

Significant evidence for small study effects was found in both the high- compared with the low-intake (P < 0.01) and the dose-response analyses (P = 0.096). Visual inspection of the funnel plots (dose-response analysis) suggests little symmetry (**Supplemental Figure 8**). Evidence exists of a nonlinear dose-response association (P-nonlinearity < 0.001; n = 14 studies). The risk of mortality decreased by 11% with increasing intake of vegetables up to ∼300 g/d. No benefit was apparent when increasing intake above this value (Figure 2).

Fruit

Thirty-four studies with 120,033 mortality cases were included in the high- compared with the low-intake meta-analysis (overall intake range: 0−626 g/d). An inverse association was observed (RR: 0.91; 95% CI: 0.89, 0.94; I^2 = 77%; P-heterogeneity < 0.001) (**Supplemental Figure 9**). Each additional daily 100 g of fruits was inversely associated with mortality risk (RR: 0.94; 95% CI: 0.92, 0.97; I^2 = 82%; P-heterogeneity < 0.001; n = 17) (**Supplemental Figure 10**).

The heterogeneity persisted largely in additional analyses stratified by sex, follow-up duration, geographic location, number of cases, and dietary assessment (**Supplemental Table 17**). The inverse association was not observed in studies from the United States or studies including only women. However, no evidence of heterogeneity was detected between subgroups in stratified analyses.

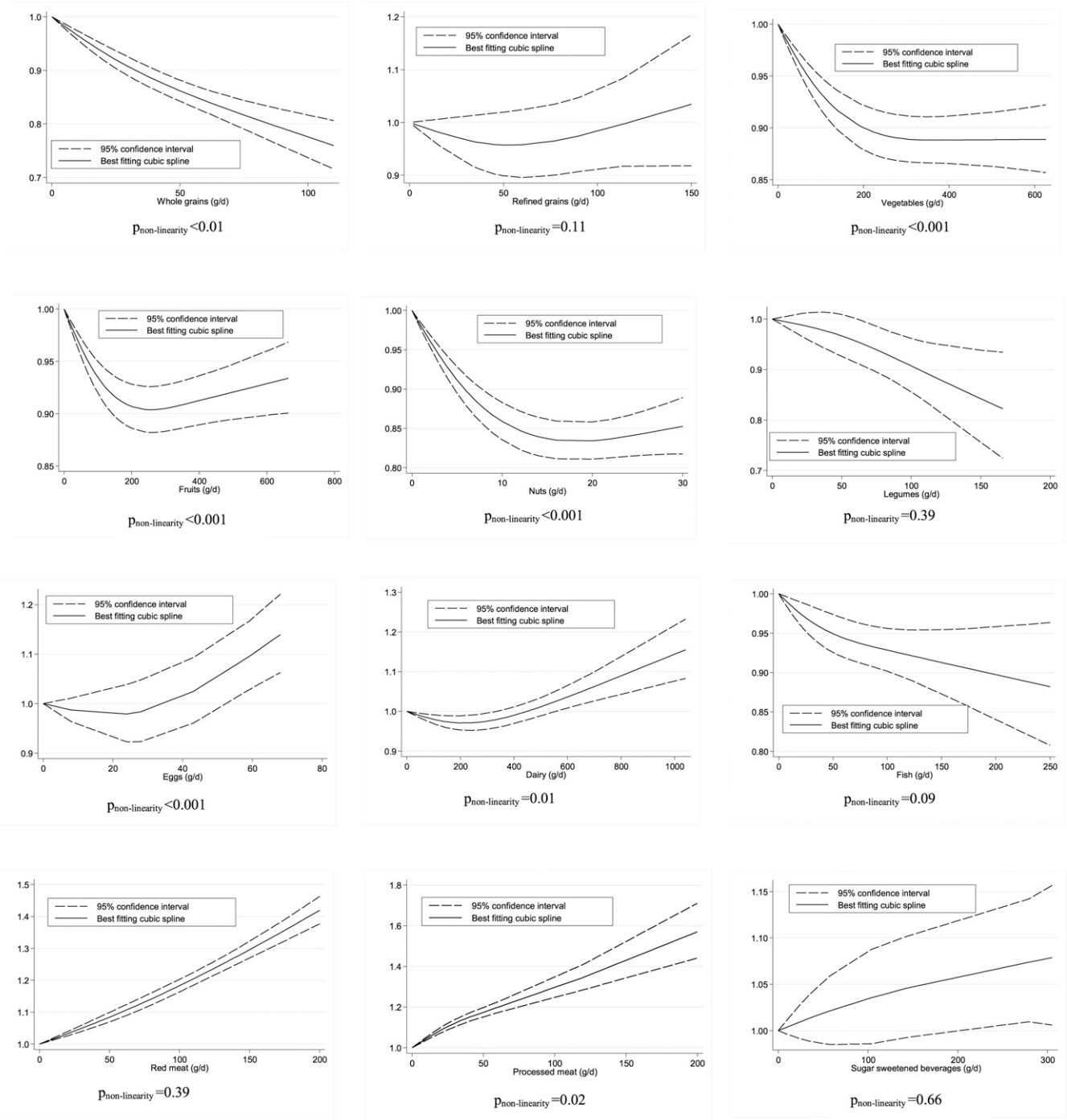

FIGURE 2 Nonlinear dose-response relation between daily intakes of whole grains, refined grains, vegetables, fruits, nuts, legumes, eggs, dairy, fish, red meat, processed meat, and sugar-sweetened beverages and risk of all-cause mortality.

Significant evidence was found for small study effects in the high- compared with the low-intake ($P < 0.001$) and dose-response analyses ($P < 0.001$). Visual inspection of the funnel plots (dose-response analysis) suggests little symmetry (**Supplemental Figure 11**). Evidence exists of a nonlinear dose-response association (P-nonlinearity < 0.001; $n = 13$ studies). The risk of all-cause mortality decreased by ~10%, with increasing intake of fruit up to ~250–300 g/d. No benefit was apparent when increasing intake above this value (Figure 2).

Nuts

Sixteen studies with 80,204 mortality cases were included in the high- compared with the low-intake meta-analysis (overall intake range: 0−52 g/d). A strong inverse association was observed for the highest compared with the lowest nut intake category (RR: 0.80; 95% CI: 0.74, 0.86; $I^2 = 84\%$; P-heterogeneity < 0.001) (**Supplemental Figure 12**), and for each additional daily 28 g (RR: 0.76; 95% CI: 0.69, 0.84; $I^2 = 82\%$; P-heterogeneity < 0.001; $n = 16$) (**Supplemental Figure 13**).

The strong inverse association and heterogeneity observed persisted in additional stratified analyses (**Supplemental Table 18**). Evidence was seen of heterogeneity between subgroups stratified by follow-up duration and geographic location, showing stronger inverse associations in European and Asian studies, and in studies with a shorter-term follow-up. Significant evidence for small study effects was detected in the high- compared with the low-intake ($P < 0.001$) and dose-response analyses ($P < 0.01$). Visual inspection of the funnel plots suggests little symmetry (**Supplemental Figure 14**). Evidence exists of a nonlinear dose-response association (P-nonlinearity < 0.001; $n = 13$ studies). The risk of all-cause mortality decreased by ~17% with increasing intake of nuts up to ~15–20 g/d. No benefit was apparent when increasing intake above this value (Figure 2).

Legumes

Seventeen studies with 53,085 mortality cases were included in the high- compared with low-intake meta-analysis (overall intake range: 6−166 g/d). An inverse association was observed for the highest compared with lowest legume intake categories (RR: 0.96; 95% CI: 0.93, 1.00; $I^2 = 48\%$; P-heterogeneity = 0.01) (**Supplemental Figure 15**), but not for each additional daily 50 g (RR: 0.96; 95% CI: 0.90, 1.01; $I^2 = 48\%$; P-heterogeneity = 0.09; $n = 6$) (**Supplemental Figure 16**).

We observed an inverse association for studies conducted in Asia and Australia, and studies with long-term follow-up (**Supplemental Table 19**). We found evidence of heterogeneity between subgroups stratified by geographic location. No evidence for small study effects was detected in the high- compared with low-intake analysis ($P = 0.32$). No evidence of a nonlinear dose-response association was observed (P-nonlinearity = 0.39; $n = 4$ studies). The risk of all-cause mortality decreased by ~16% with increasing intake of legumes up to ~150 g/d (Figure 2).

Eggs

Eight studies with 30,352 mortality cases were included in the analysis of the highest compared with the lowest intake category (overall intake range: 4−68 g/d). A positive association was observed for the highest compared with the lowest egg intake category (RR: 1.06; 95% CI: 1.00, 1.12; $I^2 = 71\%$; P-heterogeneity < 0.001) (**Supplemental Figure 17**), and for each additional daily 50 g (RR: 1.15; 95% CI: 0.99, 1.34; $I^2 = 87\%$; P-heterogeneity < 0.001; $n = 5$) (**Supplemental Figure 18**).

The observed heterogeneity persisted in additional analyses stratified by sex, follow-up duration, geographic location, and number of cases. We observed a positive association for studies of men, short-term studies, and studies applying a validated dietary assessment (**Supplemental Table 20**). Evidence of heterogeneity between subgroups was detected for follow-up duration and dietary assessment. Moreover, significant positive associations were observed for studies with ≥1000 mortality cases. We found evidence of a nonlinear dose-response association (P-nonlinearity < 0.001; $n = 3$ studies). The risk of all-cause mortality increased by ~10% with increasing intake of eggs up to ~60 g/d (Figure 2).

Dairy

Twenty-seven studies with 126,759 mortality cases were included in the meta-analysis of the highest compared with the lowest intake category (overall intake range: 0−1041 g/d). No association was observed for the high compared with the low intake (RR: 1.03; 95% CI: 0.98, 1.07; $I^2 = 94\%$; P-heterogeneity < 0.001) (**Supplemental Figure 19**), or for each additional daily 200 g of dairy products (RR: 0.98; 95% CI: 0.93, 1.03; $I^2 = 96\%$; P-heterogeneity < 0.001; $n = 16$) (**Supplemental Figure 20**).

The observed heterogeneity persisted in additional analyses stratified by sex, duration of follow-up, geographic location, and number of cases. In subgroup analyses no significant difference comparing low-fat and high-fat dairy products was observed (**Supplemental Table 21**). Some evidence of heterogeneity between subgroups in stratified analyses was observed (number of cases).

We found evidence for small study effects in the dose-response meta-analysis ($P = 0.09$), but not in the high- compared with the low-intake analysis ($P = 0.59$). Visual inspection of the funnel plots (dose-response analysis) suggests moderate symmetry (**Supplemental Figure 21**). Evidence of a nonlinear dose-response association was seen between dairy products and all-cause mortality (P-nonlinearity = 0.01; $n = 12$ studies). No detrimental effects were observed up to an intake of ~750 g/d, whereas intakes of ≤1000 g/d were associated with a 15% increased risk of mortality (Figure 2).

Fish

Thirty-nine studies with 157,688 mortality cases were included in the meta-analysis of the highest compared with the lowest intake category (overall intake range: 0−225 g/d). An inverse association was observed for the highest compared with the lowest fish intake category (RR: 0.95; 95% CI: 0.92, 0.98; $I^2 = 51\%$; P-heterogeneity < 0.001) (**Supplemental Figure 22**), and for each additional daily 100 g (RR: 0.93; 95% CI: 0.88, 0.98; $I^2 = 53\%$; P-heterogeneity < 0.01; $n = 19$) (**Supplemental Figure 23**).

We observed statistically significant heterogeneity in subgroups stratified for geographic location but no significant associations for studies conducted in Europe (**Supplemental Table 22**). No evidence for small study effects was observed ($P = 0.30$), and visual inspection of the funnel plots suggests symmetry (**Supplemental Figure 24**). We found no evidence of a nonlinear dose-response association (P-nonlinearity = 0.09; $n = 19$ studies). The risk decreased by 10% with increasing intake of ≤200 g fish/d (Figure 2).

Red meat

Twelve studies with 177,655 mortality cases were included in the high- compared with low-intake meta-analysis (overall intake range: 0−200 g/d). A positive association was observed (RR: 1.10; 95% CI: 1.00, 1.22; $I^2 = 93\%$; P-heterogeneity < 0.001) (**Supplemental Figure 25**). Each additional daily 100 g of red meat was positively associated with risk of all-cause mortality (RR: 1.10; 95% CI: 1.04, 1.18; $I^2 = 92\%$; P-heterogeneity < 0.001; $n = 10$) (**Supplemental Figure 26**).

The observed positive associations and heterogeneity persisted in additional analyses stratified by sex, follow-up duration, geographic location, and number of cases. We observed a positive association for studies of men, those conducted in the United States, long-term studies, and studies including ≥1000 mortality cases (**Supplemental Table 23**). Evidence of heterogeneity between subgroups in stratified analyses was detected for geographic location and number of cases.

We found evidence of small study effects in the dose-response analysis ($P = 0.051$) and the analysis of high compared with low intake ($P = 0.08$). Visual inspections of the funnel plots suggest low symmetry (**Supplemental Figure 27**). No evidence of a nonlinear dose-response association was found (P-nonlinearity = 0.30; $n = 10$ studies) (Figure 2).

Processed meat

Seven studies with 143,572 mortality cases were included in the high- compared with the low-intake meta-analysis (overall intake range: 0−200 g/d). A positive association was observed (RR: 1.21; 95% CI: 1.16, 1.26; $I^2 = 56\%$; P-heterogeneity = 0.03) (**Supplemental Figure 28**). Each additional daily 50 g of processed meat was associated with a risk of all-cause mortality (RR: 1.23; 95% CI: 1.12, 1.36; $I^2 = 94\%$; P-heterogeneity < 0.001; $n = 7$) (**Supplemental Figure 29**). The observed positive associations and heterogeneity persisted in additional analyses stratified by sex, follow-up duration, geographic location, and number of cases (**Supplemental Table 24**). We detected evidence of heterogeneity in stratified analyses for geographic location, showing stronger associations in studies from the United States compared with Europe.

Although the test for nonlinearity was significant (P-nonlinearity = 0.02; $n = 7$ studies), a clear dose-response relation was observed. The risk of all-cause mortality increased by ∼60% with increasing intake of processed meat up to ∼200 g/d (Figure 2).

Sugar-sweetened beverages

Five studies with 81,407 mortality cases were included in the high- compared with the low-intake meta-analysis (overall intake range: 0−930 mL/d). No association between all-cause mortality and SSBs was observed in the analysis of high compared with low intake (RR: 1.02; 95% CI: 0.97, 1.06; $I^2 = 78\%$; P-heterogeneity < 0.01) (**Supplemental Figure 30**) or the linear dose-response meta-analysis (RR: 1.03; 95% CI: 0.91, 1.18; $I^2 = 71\%$; P-heterogeneity = 0.02; $n = 4$) (**Supplemental Figure 31**). We observed a positive association in studies conducted in America (**Supplemental Table 25**). Some evidence of heterogeneity between subgroups in stratified analyses (follow-up duration and geographic location) was observed, indicating positive associations only for studies conducted in the United States and those with a shorter follow-up. We found no evidence of a nonlinear dose-response association (P-nonlinearity = 0.66; $n = 3$ studies). The risk of all-cause mortality increased by ∼7% with increasing intake of SSBs up to ∼250 mL/d (Figure 2).

Summary across food groups

Table 1 shows the RR for all-cause mortality from nonlinear dose-response analysis of the 12 food groups according to servings per day. Optimal consumption (the smallest serving with significant results and no further substantial change in risk or no further data for larger amounts) of risk-decreasing foods [3 servings whole grains/d (RR = 0.79), 3 servings vegetables/d (RR = 0.89), 3 servings fruit/d (RR = 0.90), 1 serving nuts/d (RR = 0.85), 1 serving legumes/d (RR = 0.90), and 2 servings fish/d (RR = 0.90)] results in a 56% reduction (calculated by $1 - RR_{\text{reduced}}$) compared with no consumption of these foods. The highest reduction in risk for all-cause mortality in terms of servings could be observed for whole grains: 90 g/d (3 servings/d) was associated with a 21% reduction in risk compared with no consumption of this food group. An ∼15% risk reduction was observed for 1 serving nuts/d. Furthermore, Table 1 shows that increasing the daily consumption of food with an inverse relation to risk of all-cause mortality beyond 3 servings/d of vegetables and of fruits (∼250 g/d for both), and one-half serving nuts/d (∼15 g/d) will not further reduce risk (Figure 2). We could also calculate that a consumption of risk-increasing food—2 servings red meat/d (170 g; RR = 1.35), 4 servings processed meat/d (120 g; RR = 1.35), 1 serving eggs/d (55 g; RR = 1.07), and 1 serving SSBs/d (250 mL; RR = 1.07)—is associated with a 2-fold increased risk ($RR_{\text{increased}}$) compared with no consumption. Not consuming these foods would reduce the risk of all-cause mortality by about 52% [calculated by $1 - (1 \div RR_{\text{increased}})$]. Because lower intakes of dairy consumption showed a reduced risk of mortality and higher intake values showed increased risk (largely driven by 2 cohort studies), we did not consider this food group in the optimal consumption calculations.

Risk of bias

The results varied little by methodologic assumption, including only studies with a low risk of bias (Supplemental Tables 14–25). Findings including studies with a low risk of bias suggest a smaller inverse association between fruits ($n = 4$) and nuts ($n = 5$) and all-cause mortality in the linear dose-response meta-analysis. Moreover, all of the studies included that investigated the association between eggs and all-cause mortality were rated with an unclear or high risk of bias.

Quality of meta-evidence

We rated the quality of meta-evidence for the 12 food groups. The NutriGrade meta-evidence rating was "very low" for eggs; "low" for refined grains, vegetables, fruits, and SSBs; "moderate" for nuts, legumes, dairy, fish, red meat, and processed meat; and "high" for whole grains (**Supplemental Table 26**).

DISCUSSION

The associations between 12 food groups defined a priori and risk for all-cause mortality were systematically assessed in this meta-analysis through comparison of extreme categories and dose-response analyses both for linear and nonlinear relations. Nine of the 12 food groups showed an association with all-cause mortality in the categorical or continuous dose-response analyses; an inverse association was present for whole grain, vegetable, fruit, nut, legume, and fish consumption, whereas a positive association was present for red meat, processed meat, egg, and SSB consumption. We found a clear indication for nonlinear relations between vegetables, fruits, nuts, and dairy with all-cause mortality. The NutriGrade tool for evaluating the meta-evidence suggested high confidence in the effect estimate for whole grains.

TABLE 1
Relative risks from nonlinear dose-response analysis of 12 predefined food groups and all-cause mortality according to servings per day[1]

	Servings per day						
Associations by food group	0	1	2	3	4	5	6
Inverse association							
Whole grains (30 g/d)	1.00	0.91 (0.89, 0.92)	0.84 (0.82, 0.86)	0.79 (0.76, 0.83)	NA	NA	NA
Vegetables (80 g/d)	1.00	0.94 (0.93, 0.96)	0.91 (0.89, 0.93)	0.89 (0.87, 0.92)	0.89 (0.87, 0.91)	0.89 (0.87, 0.91)	0.89 (0.86, 0.92)
Fruit (80 g/d)	1.00	0.94 (0.93, 0.96)	0.91 (0.89, 0.93)	0.90 (0.88, 0.93)	0.91 (0.88, 0.93)	0.92 (0.89, 0.94)	0.92 (0.89, 0.95)
Nuts (28 g/d)	1.00	0.85 (0.82, 0.89)	NA	NA	NA	NA	NA
Legumes (100 g/d)	1.00	0.90 (0.85, 0.96)	NA	NA	NA	NA	NA
Fish (100 g/d)	1.00	0.93 (0.90, 0.96)	0.90 (0.84, 0.96)	NA	NA	NA	NA
Positive association							
Eggs (55 g/d)	1.00	1.07 (1.01, 1.15)	NA	NA	NA	NA	NA
Red meat (85 g/d)	1.00	1.16 (1.14, 1.18)	1.35 (1.32, 1.38)	NA	NA	NA	NA
Processed meat (30 g/d)	1.00	1.12 (1.10, 1.14)	1.20 (1.17, 1.23)	1.28 (1.23, 1.32)	1.35 (1.28, 1.41)	NA	NA
Sugar-sweetened beverages (250 mL/d)	1.00	1.07 (1.01, 1.14)	NA	NA	NA	NA	NA
Inverse and positive association							
Dairy (200 g/d)	1.00	0.97 (0.95, 0.99)	0.99 (0.97, 1.01)	1.04 (1.01, 1.07)	1.11 (1.05, 1.17)	1.16 (1.08, 1.23)	NA
No association							
Refined grains (30 g/d)	1.00	0.96 (0.92, 1.01)	0.96 (0.90, 1.02)	0.97 (0.91, 1.05)	1.00 (0.92, 1.08)	1.03 (0.92, 1.16)	NA

[1] Values are risk ratios (95% CIs). NA, not applicable.

In agreement with this statement, this food group has been deemed important in the prevention of early death and disability (2).

Previous meta-analyses have shown findings similar to ours. These publications reported an inverse association of all-cause mortality with consumption of whole grains, fruits and vegetables, nuts, and fish (4–7); a positive association with consumption of red and processed meats (8); and no significant linear associations with consumption of refined grains and dairy products (5, 126). Mostly concentrating on a single food group, none of these meta-analyses is as comprehensive as ours, and most did not investigate nonlinear dose-response relations or the quality of meta-evidence. Finally, this is, to our knowledge, the first meta-analytic synthesis of any associations between legumes, eggs, and SSBs and all-cause mortality. The observed association between whole-grain intake and all-cause mortality is consistent with results from meta-analyses that associated whole-grain intake with reduced risk of CVDs, overall cancer, and especially colorectal cancer (5, 127). The inverse association we observed between fruit and vegetable consumption and all-cause mortality risk was similar to results from a previous meta-analysis (128). The inverse linear association between nut consumption and risk of all-cause mortality seen in this study was the strongest of all associations in the 12 food groups. This finding is consistent with the results of a recent meta-analysis (129) and umbrella review (130) considering multiple outcomes. Despite the absence of a linear association between legumes and all-cause mortality risk, the nonlinear analysis showed a 16% reduced risk of all-cause mortality when consuming up to ~150 g/d. Consistent with the conviction that fish consumption is good for health, our study showed an inverse association with risk of all-cause mortality when comparing the highest with the lowest intake category. According to a meta-analysis of 15 randomized controlled trials, consumption of marine n-3 PUFAs can lead to a 17% reduction in premature deaths (131), whereas a more recent meta-analysis showed no association between n-3 fatty acid supplementation and risk of all-cause mortality and CVD (132). Although the risk-decreasing potential of fish is well established and confirmed by the results of our meta-analysis, the unavoidable presence of environmental contaminants (133) should be also taken into account when larger amounts are consumed. Many large US cohort studies have consistently found evidence of an association between red and processed meats and an increased risk of all-cause mortality (119, 121). Analyses of the EPIC (European Prospective Investigation into Cancer and Nutrition) cohort showed a slight J-shaped positive association between red meat consumption and all-cause mortality; the lowest risk was identified among participants with low to moderate meat consumption (120). Despite not finding a linear association between SSBs and all-cause mortality risk in our study, the nonlinear analyses showed a 7% higher risk with increasing intake of SSBs up to ~250 mL/d. Furthermore, a recent meta-analysis showed a 22% increase in the risk of CVD for each additional serving of SSBs per day (134).

We are not arguing that the consumption of the investigated food groups are per se causally related to mortality, but rather that the study results and their synthesis via meta-analyses reflect underlying specific biological relations related to the etiology of chronic diseases and/or preclinical disorders and risk factors. Therefore, the end point "mortality" and even "cause-specific mortality" could be considered as important public health markers for the impact of dietary factors on the disease spectrum, and as general measures of potential disease reduction. The investigation of single food groups (or other dietary factors) with respect to mortality is based on the paradigm that compounds of the food groups can be linked to specific disease mechanisms because of their biological activity and other physiologic properties. For example, protective effects of whole grains, fruits, vegetables, nuts, and fish might be explained by anti-inflammatory, antioxidative, antiproliferative, or chemopreventive mechanisms, which have been described for a number of bioactive compounds (e.g., fiber, minerals, trace elements, vitamins, carotenoids, polyphenols, alkylresorcinols, omega-3 fatty acids)

(135, 136). On the other hand, unfavorable effects of food groups such as red and processed meats might be based on opposite effects (proinflammatory, pro-oxidative, or carcinogenic compounds) triggered by nitrosamines, iron, or SFAs (137). The detrimental effects observed for SSBs may be partly attributed to impairments of the otherwise working regulation of hunger and satiety (138).

The investigated food groups are often part of dietary patterns or diet quality indexes, such as the Mediterranean diet, vegetarian diet, or Healthy Eating indexes. A meta-analysis of nearly 5 million subjects reported an 8% reduction of overall mortality for each 2-point increase in adherence to the Mediterranean diet (139). Moreover, in a recent meta-analysis high adherence to the Healthy Eating Index was associated with a 22% lower risk of mortality (9)

Strengths and limitations

Dietary information of most of the included studies derives from food frequency questionnaires, which represent a subjective approximation of past dietary behaviors rather than an assessment of absolute intakes. Hence, our results may reveal higher accuracy than is actually available (140). Substantial heterogeneity was found with respect to the analyzed population size, follow-up duration, baseline age, and food consumption. We conducted subgroup analyses for sex, follow-up duration, geographic location, number of cases, and dietary assessment methods in order to explore high degrees of statistical heterogeneity. Overall, for most food groups, high levels of statistical heterogeneity persisted in subgroup analyses. People with a high intake of whole grains, fruits, vegetables, fish, nuts, or legumes might have different lifestyles or a different socioeconomic status from those with lower intakes, representing important confounders (141). However, our main results were confirmed by sensitivity analyses including only studies with a low risk of bias [adjusted for important lifestyle factors (smoking, physical activity, and BMI)]. Another important limitation was the indication of small study effects such as publication bias in the analyses of vegetables, fruits, nuts, dairy products, and red meat. The results of the nonlinear association between dairy and all-cause mortality should be interpreted with caution because these observations were largely influenced by 2 cohort studies showing a strong positive association (93). Among the strengths of the present meta-analysis are the a priori published systematic review protocol (14), the comprehensive literature search, and the large numbers of prospective studies, death cases, and food groups included. Furthermore, we performed different types of analyses (high compared with low intake, dose-response meta-analysis, nonlinear dose-response analysis, and subgroup and sensitivity analyses), which allowed us to detect associations where the relation was nonlinear and find an optimal consumption with the lowest risk of all-cause mortality. Finally, we assessed the quality of the studies using meta-evidence for each food group through use of the NutriGrade scoring system.

In conclusion, an optimal intake of whole grains, vegetables, fruits, nuts, legumes, and fish, as well as reduced consumption of red and processed meats and SSBs, can lead to an important decrease—by ~80%—in the relative risk of premature death when compared with intakes always from the highest risk category. To obtain a complete picture, it seems useful to extend the type of food groups and the clinical end points to be considered. We will in the future develop methods that are able to rank foods and diseases according to their contribution to the prevention of chronic diseases.

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