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Association Between Dietary Fiber and Lower Risk of All-Cause Mortality: A Meta-Analysis of Cohort Studies

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Although in vitro and in vivo experiments have suggested that dietary fiber might have beneficial effects on health, results on the association between fiber intake and all-cause mortality in epidemiologic studies have been inconsistent. Therefore, we conducted a meta-analysis of prospective cohort studies to quantitatively assess this association. Pertinent studies were identified by searching articles in PubMed and Web of Knowledge through May 2014 and reviewing the reference lists of the retrieved articles. Study-specific risk estimates were combined using random-effects models. Seventeen prospective studies (1997–2014) that had a total of 67,260 deaths and 982,411 cohort members were included. When comparing persons with dietary fiber intakes in the top tertile with persons whose intakes were in the bottom tertile, we found a statistically significant inverse association between fiber intake and all-cause mortality, with an overall relative risk of 0.84 (95% confidence interval: 0.80, 0.87; I^2 = 41.2%). There was a 10% reduction in risk for per each 10-g/day increase in fiber intake (relative risk = 0.90; 95% confidence interval: 0.86, 0.94; I^2 = 77.2%). The combined estimate was robust across subgroup and sensitivity analyses. No publication bias was detected. A higher dietary fiber intake was associated with a reduced risk of death. These findings suggest that fiber intake may offer a potential public health benefit in reducing all-cause mortality.

diet; fiber intake; meta-analysis; mortality; prospective studies

Abbreviations: CI, confidence interval; RR, relative risk.

The number of deaths from noncommunicable diseases rose by approximately 8 million between 1990 and 2010 and accounted for 2 of every 3 deaths worldwide by 2010 (1). Of chronic noncommunicable diseases, cardiovascular disease, cancer, and diabetes are the main causes of death (1). A high-quality diet that includes functional foods or functional ingredients is one of the most promising factors in primary and secondary prevention of noncommunicable diseases (2).

Dietary fiber is widely recognized as an important part of a healthy diet. It is the edible parts of plants, or similar carbohydrates, that are resistant to digestion and absorption by the small intestine. According to the current Dietary Reference Intakes recommended by the United States Department of Agriculture, adults should consume 14 g of dietary fiber per 1,000 kcal ingested, which translates into a daily intake of approximately 25 g for women and 38 g for men. The global disability-adjusted life-years attributable to a diet low in fiber rose from 13.3 million in 1990 to 16.5 million in 2010, which indicated an increased global burden of diseases caused by low dietary fiber intake (3).

Accumulating evidence indicates that a high intake of dietary fiber might decrease the risks of coronary heart disease, stroke, hypertension, diabetes, and major cancers (4–10). Extensive prospective studies have also evaluated the association between dietary fiber intake and all-cause mortality in general healthy populations (11–19) and specific disease-related populations (20–27), such as patients with breast cancer (23–26). Because the evidence from prospective studies on dietary fiber intake in relation to all-cause mortality has not yet been summarized, we conducted a meta-analysis of prospective cohort studies to quantify this association.

METHODS

Study selection

We followed standard criteria for conducting and reporting of meta-analyses of observational studies (28). We performed a comprehensive, computerized literature search through May 2014 using the following key words in PubMed and Web of Knowledge: (dietary OR diet) AND (fiber OR fibre) AND (mortality OR death). The identified publications were reviewed independently for their relevance to the research topic by 2 authors (Y.Y., L.-G.Z.). We also manually searched the reference lists of relevant publications to identify additional studies. A set of prespecified inclusion criteria was applied during the review, and discrepancies were resolved by consensus. To be included in the meta-analysis, studies had to: 1) report all-cause/total mortality as the outcome of interest, 2) be conducted in a general healthy population or a specific disease-related population, 3) use an observational, prospective cohort design, 4) present information on dietary fiber intake as the exposure of interest, and 5) provide estimates of relative risk/hazard ratio with confidence intervals or standard errors or the data necessary to calculate these.

We used the reported relative risk to measure the association between dietary fiber intake and all-cause mortality. If multiple estimates were provided, priority was given to the multivariable-adjusted risk estimates that were adjusted for the most potential confounding factors in original studies. If more than one study was conducted in the same population, the most recent report or the report with the most applicable estimates was selected for our analysis.

Data extraction

We used a standardized protocol and reporting form to abstract the following data from each publication: the first author's name, the year of publication, the country in which the study was conducted, the duration of follow-up, the age and sex of the study population, the size of the cohort, the number of deaths, the method used to assess dietary fiber intake, the categories of dietary fiber intake and the relative risks and 95% confidence intervals for all-cause mortality associated with those categories, and the covariates included for adjustment in multivariable models.

Statistical analysis

We converted the reported relative risks into a standard scale of effect to compare persons with dietary fiber intakes in the top tertile with persons whose intakes were in the bottom tertile, in essence giving an estimate per 2.18 standard-deviation units of dietary fiber intake, where 2.18 is the difference in the means of the highest and lowest tertile of the standard normal distribution. Of note, the cutoffs for the top and bottom tertiles were study specific. All studies compared persons in the top tertile with those in the bottom tertile, but the cutoffs for those tertiles varied by study. This scaling method assumed 1) a normal distribution for the total fiber intake in the study population and 2) a log-linear association with death risk over the mid-range of the baseline value of total fiber intake. When the log relative risk for the comparison of the top and bottom tertiles of dietary fiber intake was not directly available from the published report, it was estimated as 1) a scaling factor of 2.18 divided by 1.59 times the log relative risk for comparison of the top and bottom halves, 2) a scaling factor of 2.18 divided by 2.54 times the log relative risk for comparison of the top and bottom quartiles, 3) a scaling factor of 2.18 divided by 2.80 times the log relative risk for comparison of the top and bottom quintiles, or 4) a scaling factor of 2.18 times the log relative risk for a 1-standard-deviation difference in dietary fiber intake. The standard error of the log relative risk was calculated from the number of standard errors by which the reported relationship differed from 0. More details about the scaling method have been published previously (29).

To examine the associations between dietary fiber intake and all-cause mortality, we pooled the study-specific relative risk estimates for comparison of persons whose dietary fiber intakes were in the top tertile with persons whose intakes were in in the bottom tertile, as well as for every 10-g/day increase from each study. For the study by Buck et al. (23) in which only categorical results were published, we used the method proposed by Greenland and Longnecker (30) and Orsini et al. (31) to calculate the trend in relative risk per each 10-g/day increase in dietary fiber intake. We used the random-effect model proposed by DerSimonian and Laird (32) to pool the study-specific estimates, which considered both within- and between-study variation.

For the association of dietary fiber intake (top tertile vs. bottom tertile) with all-cause mortality, we conducted analyses stratified by age, sex, study population, study location, dietary assessment method, and follow-up period. We also conducted analyses stratified by whether the studies adjusted for potentially important confounders or important risk factors, including body mass index (weight (kg)/height $(m)^2$), smoking status, alcohol consumption, physical activity level, serum cholesterol level, and blood pressure. In addition, we performed a sensitivity analysis of the influence of individual studies on the summary estimate by repeating the meta-analysis excluding 1 study at a time. Heterogeneity among studies was assessed with the Q and I^2 statistics, and results were defined as heterogeneous for a P value < 0.10 or an $I^2 > 50\%$ (33). Associations that resulted from studies with small sample sizes, such as publication bias, were evaluated by visual inspection of funnel plot and formal testing by using Egger's test (34).

Statistical analyses were conducted using Stata, version 11.0 (StataCorp LP, College Station, Texas). Two-sided P values < 0.05 were considered statistically significant unless otherwise specified.

RESULTS

Literature search

Our systematic literature search yielded a total of 17 articles in which the association between dietary fiber intake and all-cause mortality was reported (11–27). A flow chart for the search is presented in Figure 1. Of the 2,661 records identified from the 2 databases, 840 records were excluded because they were duplicates. After a review of the titles

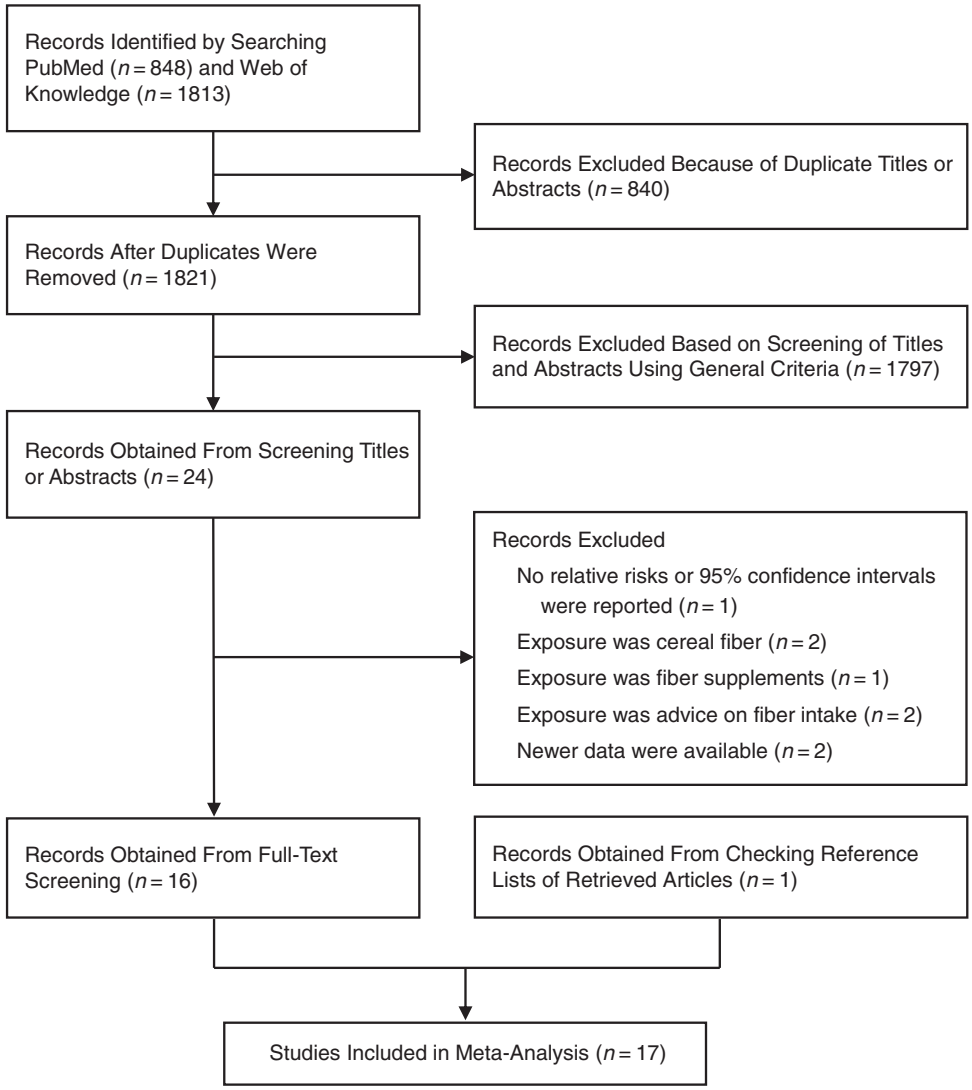

Figure 1. Flow chart detailing the search for and selection of cohort studies in the meta-analysis. Studies were published from 1964 to 2014.

and abstracts based on the prespecified inclusion criteria, 1,797 articles were further excluded. After reviewing the full text of the remaining 24 cohort studies, 8 studies were excluded because 1) no available relative risks or 95% confidence intervals were reported ($n = 1$) (35); 2) the exposures of interest were cereal fiber ($n = 2$) (36, 37), fiber supplements ($n = 1$) (38), or advice on fiber intake ($n = 2$) (39, 40); and 3) newer data were available ($n = 2$) (41, 42). One study (27) that was identified by checking the reference lists of retrieved articles was also included, giving a total of 17 studies in the final analysis (11–27).

Study characteristics

Descriptive data for the studies included in our analysis are summarized in Web Table 1 (available at http://aje.oxfordjournals.org/). There were a total of 67,260 deaths among 982,411 participants in the 17 cohort studies. Of the included studies, 9 were conducted in the general healthy population (11, 13–19, 22), 4 were conducted in breast cancer patients (23–26), and 4 were conducted in other disease-related populations (1 in patients with gastric cancer (27), 1 in patients with type 1 diabetes (21), 1 in survivors of myocardial infarction (20), and 1 in patients with chronic kidney disease (22)). The included studies were conducted in the United States ($n = 7$) (14, 16, 20, 22, 24–26), Europe ($n = 9$) (11–13, 15, 18, 19, 21, 23, 27), and Asia ($n = 1$) (17). Most of the included studies used food frequency questionnaires to assess dietary fiber intake ($n = 12$) (12–14, 17–20, 23–27), and the others used 24-hour dietary recall ($n = 2$) (16, 22), 3-day dietary record ($n = 1$) (21), self-administered quantitative dietary questionnaires ($n = 1$) (11), and cross-checked dietary history methods ($n = 1$) (15). All of the included studies adjusted for age, and most of them included adjustment for

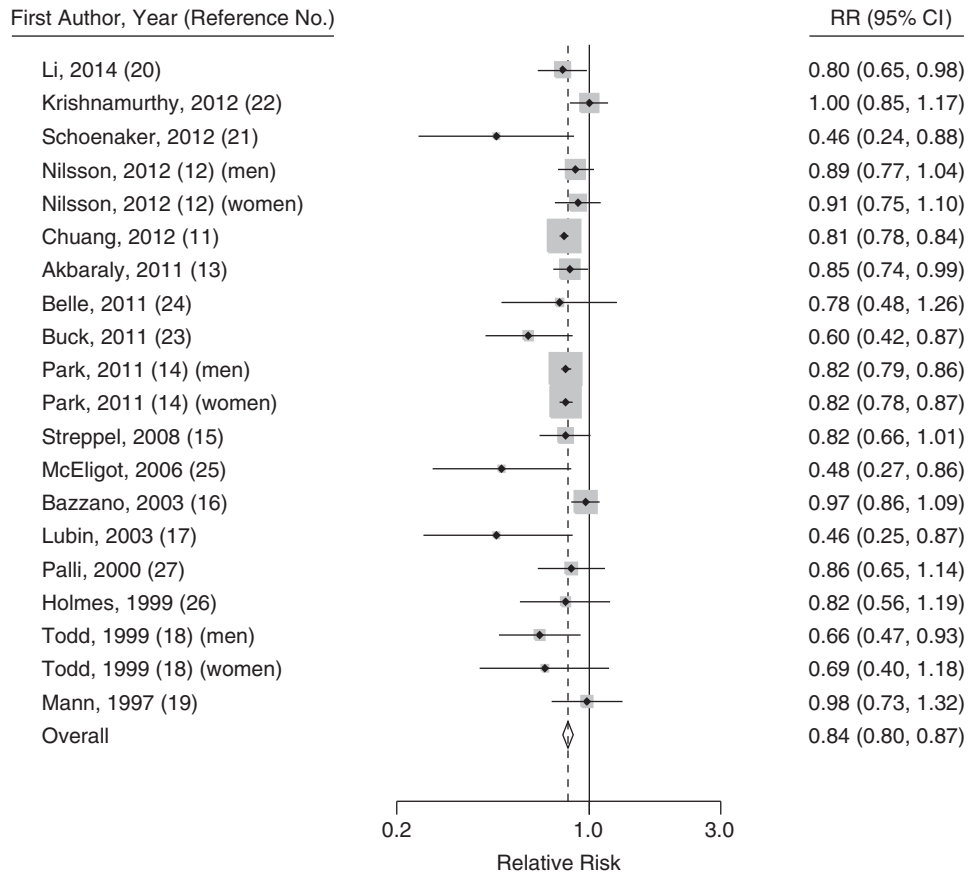

Figure 2. Results from meta-analysis of the association between dietary fiber intake and all-cause mortality (random-effects model), 1997–2014. Relative risks (RRs) compare persons in the top and bottom tertiles of dietary fiber intake. Squares represent study-specific estimates (size of the square reflects the study-specific statistical weight); horizontal lines represent 95% confidence intervals (CIs); diamonds represent the summary estimate with corresponding 95% confidence interval.

other most likely potential confounders, such as sex (if available), body mass index, total energy intake, smoking status, and alcohol drinking.

Overall analyses

Figure 2 shows the relative risks of all-cause mortality for comparison of persons with dietary fiber intake levels in the top tertile and with persons with intakes in the bottom tertile for 17 prospective cohort studies. The pooled relative risk showed a 16% reduction in the risk for persons with dietary fiber intake levels in the top tertile compared with those with intakes in the bottom tertile (relative risk (RR) = 0.84; 95% confidence interval (CI): 0.80, 0.87). Low statistical heterogeneity was detected in the study results ($P = 0.029$; $I^2 = 41.2\%$). There was no evidence of publication bias in the 17 studies, as tested using Egger's test ($P = 0.610$; Web Figure 1). Figure 3 shows the relative risks of all-cause mortality for the study-specific estimate per each 10-g/day increase in fiber intake for 8 cohort studies. There was a 10% reduction in risk for each 10-g/day increase in dietary fiber, with significant heterogeneity (RR = 0.90; 95% CI: 0.86, 0.94; $I^2 = 77.2\%$; P for heterogeneity < 0.001).

Subgroup and sensitivity analyses

Table 1 shows the pooled relative risks of all-cause mortality when comparing persons in the top tertile of dietary fiber intake with those in the bottom tertile in strata of selected study characteristics. We detected a significant heterogeneity between the subgroup stratified by adjustment for serum cholesterol level ($P_h = 0.016$), which indicated that the association between dietary fiber intake and all-cause mortality differed depending on whether studies had adjusted for blood cholesterol level. In the 14 studies that did not adjust for serum cholesterol level, the pooled relative risk was 0.82 (95% CI: 0.79, 0.84; $I^2 = 11.3\%$), with little heterogeneity (P for heterogeneity = 0.324). The inverse association was attenuated when the analysis was restricted to the 3 studies that adjusted for cholesterol level (RR = 0.90; 95% CI: 0.77, 1.06; $I^2 = 51.7\%$; P for heterogeneity = 0.102). Moreover, compared with the pooled relative risk for the association of

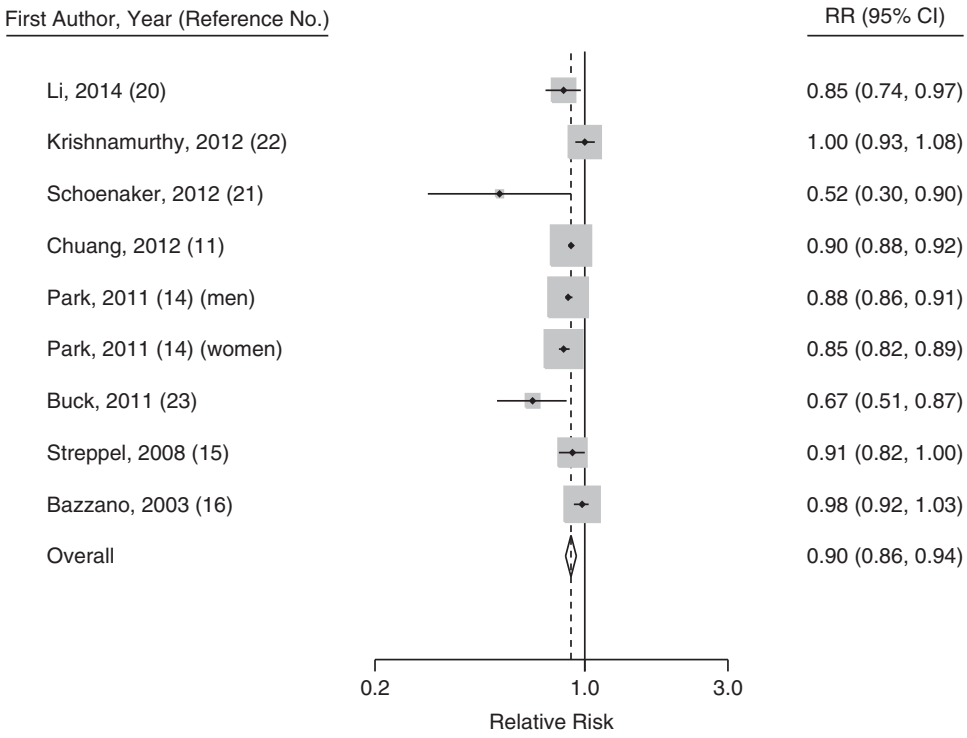

Figure 3. Results from meta-analysis of the association between each 10-g/day increase in dietary fiber intake and all-cause mortality (random-effects model), 2003–2014. Squares represent study-specific estimates (size of the square reflects the study-specific statistical weight); horizontal lines represent 95% confidence interval (CIs); diamonds represent the summary estimate with corresponding 95% confidence interval. RR, relative risk.

dietary fiber intake with all-cause mortality among a general healthy population and other disease-related populations, the inverse association was much stronger when the analysis was restricted to the 4 studies conducted in patients with breast cancer (RR = 0.68; 95% CI: 0.54, 0.84; $I^2 = 3.2\%$; P for heterogeneity = 0.376) (23–26). However, we did not detect significant heterogeneity by different study population between subgroups (P for heterogeneity = 0.228). We also did not detect significant heterogeneity between the other subgroups, such as different dietary assessment methods (P for heterogeneity = 0.307), or by categories of follow-up time (P for heterogeneity = 0.286). For studies in which dietary fiber intake was assessed using a food frequency questionnaire (12–14, 17–20, 23–27), the pooled relative risk was 0.82 (95% CI: 0.79, 0.85; $I^2 = 9.4\%$; P for heterogeneity = 0.348), which was slight lower than that for persons assessed using other dietary assessment methods (RR = 0.87; 95% CI: 0.77, 0.99; $I^2 = 76.1\%$; P for heterogeneity = 0.002) (11, 15, 16, 21, 22). For studies with a median follow-up time of 10 years or less (14, 18, 20–25, 27), the pooled relative risk was 0.81 (95% CI: 0.76, 0.87; $I^2 = 42.0\%$; P for heterogeneity = 0.069), which was slight lower than that for studies with a median follow-up greater than 10 years (RR = 0.86; 95% CI: 0.80, 0.93; $I^2 = 45.9\%$; P for heterogeneity = 0.063) (11–13, 15–17, 19, 26).

In sensitivity analyses, we sequentially excluded 1 study at a time to recalculate the pooled relative risks of all-cause mortality when comparing persons in the top tertile of dietary fiber intake with those in the bottom tertile. The 16 pooled relative risks for sensitivity analyses were still statistically significant and similar to the overall estimate (data not shown). When we excluded 1 study that did not adjust for or consider total energy intake as a confounder in the model (19), the result was not altered materially.

DISCUSSION

To our knowledge, the present study is the first meta-analysis in which the association between dietary fiber intake and all-cause mortality has been quantitatively assessed. In our meta-analysis, increased dietary fiber intake was significantly associated with a reduced risk of death. The combined estimate for all-cause mortality was robust across subgroup and sensitivity analyses, and no publication bias was detected.

Dietary fiber intake might lower all-cause mortality rates by decreasing the risk of major chronic diseases, including cardiovascular disease and major cancers, which are notorious as the major killers worldwide (4, 8–10). Findings from the present meta-analysis are in agreement with those from previous meta-analyses of dietary fiber intake in relation to the risk of chronic diseases. Summary results based on 22 prospective studies showed that the risks of cardiovascular disease and coronary heart disease both decreased by 9% for each 7-g/day increase in dietary fiber intake (4). In another

Table 1. Risk Ratios for All-Cause Mortality Comparing Persons With Dietary Fiber Intake Levels in the Top Tertile With Persons With Intakes in Bottom Tertile, by Study Characteristics, 1997–2014

Subgroup by Study Characteristic	No. of Studies	Pooled RR	95% CI	I^2, %	*P* for heterogeneity[a]	*P* for heterogeneity[b]
Mean or median age, years						0.124
<54	8	0.87	0.80, 0.95	58.7	0.010	
≥54	7	0.80	0.76, 0.85	25.1	0.228	
Sex						0.623
Female	9	0.83	0.79, 0.86	4.1	0.401	
Male	6	0.80	0.76, 0.85	40.6	0.135	
Study population						0.228
General healthy population	9	0.84	0.80, 0.87	35.7	0.105	
Breast cancer patients	4	0.68	0.54, 0.84	3.2	0.376	
Other disease-related population[c]	4	0.85	0.69, 1.03	57.8	0.069	
Geographic location						0.524
United States	7	0.85	0.80, 0.92	56.0	0.026	
Europe	9	0.82	0.78, 0.87	17.8	0.274	
Dietary assessment method						0.307
Food frequency questionnaire	12	0.82	0.79, 0.85	9.4	0.348	
Other[d]	5	0.87	0.77, 0.99	76.1	0.002	
Median follow-up time, years						0.286
≤10	9	0.81	0.76, 0.87	42.0	0.069	
>10	8	0.86	0.80, 0.93	45.9	0.063	
Potential confounders or risk factors[e]						
Body mass index[f]						0.104
Yes	13	0.83	0.79, 0.86	32.1	0.105	
No	4	0.90	0.74, 1.10	47.2	0.128	
Smoking status						0.203
Yes	14	0.84	0.81, 0.88	44.0	0.030	
No	3	0.71	0.57, 0.89	18.7	0.297	
Alcohol consumption						0.848
Yes	12	0.84	0.80, 0.88	48.9	0.017	
No	5	0.85	0.74, 0.96	14.0	0.325	
Physical activity level						0.367
Yes	11	0.83	0.79, 0.87	52.9	0.013	
No	6	0.87	0.80, 0.95	0	0.498	
Serum cholesterol level						0.016
Yes	3	0.90	0.77, 1.06	51.7	0.102	
No	14	0.82	0.79, 0.84	11.3	0.324	
Blood pressure						0.073
Yes	6	0.85	0.75, 0.96	55.8	0.035	
No	11	0.82	0.79, 0.84	10.0	0.345	

Abbreviations: CI, confidence interval; RR, relative risk.

[a] *P* value for heterogeneity within each subgroup.

[b] *P* value for heterogeneity between subgroups in meta-regression analysis.

[c] Other disease-related populations included patients with gastric cancer ($n = 1$), type 1 diabetes ($n = 1$), or chronic kidney disease ($n = 1$), as well as survivors of myocardial infarction ($n = 1$).

[d] Other dietary assessment methods included 24-hour dietary recall ($n = 2$), 3-day dietary record ($n = 1$), self-administered quantitative dietary questionnaires ($n = 1$), and cross-checked dietary history ($n = 1$).

[e] The subgroups were classified according to whether the reported risk estimates in the studies included adjustment for the variable indicated.

[f] Weight (kg)/height $(m)^2$.

meta-analysis of stroke, each 7-g/day increase in dietary fiber intake was associated with a 7% decrease in the risk of hemorrhagic plus ischemic stroke (5). Among cancer types, cancers of the breast, colorectum, and stomach have been most consistently associated with dietary fiber intake (8–10). The 3 most recent meta-analyses showed that every 10-g/day increase in dietary fiber intake was associated with 5%, 10%, and 44% decreases in the risks of cancers of breast, colorectum, and stomach, respectively (8–10). These figures from the meta-analyses support our finding that increased intake of dietary fiber is associated with a lower overall risk of death.

An inverse association between dietary fiber intake and all-cause mortality is biologically plausible. Dietary fiber has been shown to 1) inhibit cholesterol synthesis and reduce serum cholesterol levels by increasing the production of short-chain fatty acid and the rate of bile excretion; 2) lower blood pressure; 3) promote body-weight loss by regulating energy intake; 4) slow glucose absorption and improve insulin sensitivity; 5) reduce contact time between potential carcinogens and mucosal cells by increasing fecal bulking and viscosity; 6) aid in the binding between bile acids and carcinogens; 7) improve the amount of estrogen excreted in the feces through an inhibition of estrogen absorption in the intestines; and 8) increase levels of antioxidants (43, 44). All of these biological effects may be related to a lower risk of some major chronic diseases, such as cardiovascular disease, and major cancers, such as breast cancer and colorectal cancer (43, 44).

Most studies included in the present meta-analysis were adjusted for at least some of the major potential confounders or risk factors, such as age, physical activity level, smoking status, alcohol consumption, total energy intake, and other dietary factors. When we restricted the analysis to studies that were adjusted for potential confounders or risk factors, the magnitude of the associations in the subgroups were similar to those of the overall association, except for the subgroup that included adjustment for serum total cholesterol level. Dietary fiber intake could influence the risk of all-cause mortality via several different mechanisms. Controlling for any of the intermediate variables, such as total cholesterol levels, in the hypothesized casual pathway between dietary fiber and all-cause mortality might lead to overadjustment and thus bias the result towards null (45). Therefore, the true association between dietary fiber intake and all-cause mortality may be even stronger.

Another issue is misclassification of dietary fiber intake due to the self-reported nature of the exposure in the included studies. In cohort studies that use more than 2 categories of exposure (or a continuously assessed exposure), it would be difficult to assess the direction of the bias even if nondifferential misclassification occurred. Moreover, potential systematic measurement error might occur because some sort of underlying diseases might lower dietary fiber intake. This kind of systematic error is hard to avoid and therefore might bias the results in most of the observational studies with dietary factors as the exposure of interest. Of note, this kind of bias could be partly reflected in subgroup results stratified by follow-up time, because persons with underlying diseases tend to die earlier and thus may have shorter follow-up times than participants who remain healthy after the baseline survey. In the subgroup analysis stratified by follow-up time, for studies with a median follow-up time of 10 years or less, the pooled relative risk was just slightly lower than for those with a median follow-up time greater than 10 years. Thus, even if this kind of bias does exist, it might only have a small effect on the final risk estimate.

Moreover, the range of dietary fiber intakes between the highest and lowest categories varied between studies, and the risk estimates would be assumed to be higher in studies with broader ranges of fiber intake. Thus, we did not pool the study-specific relative risk estimates for the highest versus lowest intake categories. Instead, we converted the reported relative risk estimates onto a standard scale of effect, comparing persons in the top tertile of dietary fiber intake with those in the bottom tertile so that the same categories of intake were compared with one another. This scaling method would be a useful approach to lower the heterogeneity between studies. Of note, the heterogeneity was reduced by 10%, from 51.3% (highest vs. lowest intake) (data not shown) to 41.2% (top tertile vs. bottom tertile). However, there was still low heterogeneity among study results (different strengths of associations) in present meta-analysis. There are several potential explanations for the observed heterogeneity. First, the study populations were different between studies. In subgroup analysis by study population, women with breast cancer were observed to benefit more from increasing their dietary fiber intake than were persons in the general healthy population, but we did not detect a significant heterogeneity between subgroups stratified by study population. Second, the sources and types of dietary fiber included in the intake groups differed. Dietary fiber is plentiful in fruits, vegetables, and cereal and includes both soluble and insoluble fiber. Total fiber intake in each of the included studies, or even within a specific study conducted in various study areas, might come from different sources and types and thus have different strengths of association with all-cause mortality. For example, in European Prospective Investigation Into Cancer and Nutrition cohort (EPIC) (11), the main sources of total dietary fiber intake differed across countries; the strongest associations were observed in the Danish and Greek cohorts, which also had the highest percentages of fiber from cereals and vegetables (56% and 16%, respectively, in Denmark and 29% and 36%, respectively, in Greece). Third, the size of cohort and the length of follow-up varied from study to study. For example, for the included studies in this meta-analysis, the cohort size ranged from 382 participants in the study by Palli et al. (27) to 452,717 participants in the study by Chuang et al. (11). The length of follow-up ranged from 6.4 years in the study by Buck et al. (23) to 40 years in the study by Streppel et al. (15). In subgroup analysis, the slight different strengths of association by follow-up time could have several implications. First, the aforementioned bias caused by certain types of underlying diseases in the baseline survey might occur in the first several years of follow-up. Second, all of the included studies assessed dietary fiber intake at the baseline surveys, which were years before the occurrence of end point. Participants could have changed their dietary habits, such as their intakes of dietary fiber, during a long period of follow-up. Thus, compared with studies with a follow-up time of 10 years or less, there is higher possibility that the exposure misclassification occurred for studies with a follow-up time

greater than 10 years when only the baseline measurements were used. Third, the critical window of effect for dietary fiber might play an important role in total deaths. The protective effect of dietary fiber could be stronger in the first several years and tend to decline increasing with follow-up time.

A strength of the present meta-analysis was the prospective cohort design of the included studies, which should have greatly reduced the potential for the selection and recall bias. The large number of total participants (982,411 men and women) and deaths (67,260 deaths) provided sufficient statistical power to quantitatively assess the relationship between dietary fiber intake and all-cause mortality. Most of the studies included in the meta-analysis adjusted for large numbers of major confounders. Because of this, the present pooled estimate may be less likely to be biased.

Our study also has some limitations. First, because of the observational design, residual confounding effects might distort the association between dietary fiber intake and all-cause mortality, and we were not able to address problems with confounding that were inherent in the original studies. For example, the inverse association between dietary fiber intake and all-cause mortality could be attributed to other factors related to fiber intake, such as vegetable and fruit intakes, physical exercise level, or other healthy habits and dietary factors. Second, publication bias might have influenced the results. Although there is no evidence of publication bias in the present meta-analysis, tests for publication bias have low statistical power, especially when the number of studies is limited. Third, although many studies indicated a positive association of dietary fiber intake with all-cause mortality, the strengths of the associations differed between studies, which resulted in statistical heterogeneity. The heterogeneity might come from various sources, and it somewhat limits the interpretation of the results. Although the scale method could help to lower the heterogeneity between the studies, the fact that the studies did not compare the same absolute intake levels could have influenced our results. In addition, most of the studies in the present meta-analysis were conducted in Western countries. The 1 study that was conducted in Israeli had a relatively lower relative risk of 0.46 (95% CI: 0.25, 0.87) for persons with dietary fiber intake levels in the top tertile versus persons with intakes the bottom tertile. This may somewhat limit the generalizability of the results from our meta-analysis.

In conclusion, in the present meta-analysis, we demonstrated that an increased fiber intake is associated with a reduced risk of death. These findings add to and extend the evidence that increased fiber intake may exert healthy effects and decrease the risk of all-cause mortality. Thus, one should be encouraged to increase his/her dietary fiber intake to potentially decrease the risk of premature death.

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