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META-ANALYSIS

Dietary fiber consumption and risk of stroke

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Abstract Observational studies suggest an association between dietary fiber consumption and risk of stroke, but the results are inconclusive. The authors conducted a meta-analysis of prospective cohort studies to evaluate the relation between dietary fiber consumption and stroke risk and mortality. Relevant studies were identified by searching PubMed, Embase, and ISI Web of Science through February 2013. We included prospective studies that reported relative risks (RRs) with 95 % confidence intervals (CIs) for the association between dietary fiber consumption and stroke risk and mortality. Both fixed- and random-effects models were used to calculate the summary risk estimates. Eleven prospective studies involving 325,627 participants were included in the meta-analysis. The pooled RR of stroke for the highest compared with the lowest dietary fiber consumption was 0.83 (95 % CI 0.74, 0.93). In addition, the increment in dietary fiber consumption was associated with decreased stroke risk in a dose–response manner. Sensitivity analysis restricted to studies with control for conventional risk factors yielded similar results, and omission of any single study had little effect on the combined risk estimate. Moreover, there was a trend toward an inverse association between higher fiber consumption and stroke mortality (RR 0.85; 95 % CI 0.60, 1.20), although it is not significant. This meta-analysis indicated that dietary fiber consumption is inversely associated with stroke risk, and the effect is probably independent of conventional risk factors. Our results support recommendations for higher consumption of fiber-rich foods to prevent stroke.

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Introduction

Stroke is a common neurological disease and a leading cause of death and long-term disability worldwide [1]. Lifestyle factors play an important role in the prevention of stroke, among which dietary fiber has received considerable interest [2, 3]. Dietary fiber may reduce the risk of stroke through a variety of mechanisms, such as lowering blood pressure [4], improving blood lipid profiles [5], and improving insulin sensitivity [6], which may prevent or delay development of atherosclerosis. Dietary fiber consumption in the United States and European countries is around 15 g/day, which is only half the recommended amount [7].

In recent years, many prospective cohort studies investigated the association between dietary fiber consumption and risk of stroke [8–18]. However, the results of these studies remain conflicting rather than conclusive, with most showing a weak or null association. Although a previous meta-analysis [19] reported a significant relation between dietary fiber consumption and stroke risk, evidence was limited because only 6 cohort studies were included. Of note, there was no subgroup analysis by fiber source (cereal, fruit and vegetable) and fiber type (soluble and insoluble) in the previous meta-analysis. Because different sources and types of fiber may have varying effects in stroke prevention, investigating the source and type differences in the fiber consumption-stroke association is of interest. In addition, the association between fiber consumption and stroke mortality is still unknown. Moreover, whether fiber consumption is an independent protective factor or only a silent marker of stroke remains unclear. An improved understanding of this topic may have

important clinical and public health implications given the possibility that increasing fiber consumption in the general population might reduce the incidence of stroke events.

Therefore, we carried out a meta-analysis of prospective cohort studies to summarize the epidemiologic evidence on the association between dietary fiber consumption and stroke risk and mortality.

Methods

Literature search and selection

We performed a systematic search of PubMed, Embase, and ISI Web of Science through February 2013 by using the keywords dietary fiber combined with stroke, cerebrovascular disease, cerebrovascular disorder, cerebrovascular accident, cerebral infarction, and cerebral hemorrhage. Moreover, the references of identified publications and recent reviews were searched for further potentially relevant articles. Only published studies in humans were included. No language restrictions were imposed. Our study was designed, conducted, and reported in adherence to standards of quality for reporting meta-analyses [20].

Studies included in this meta-analysis have to meet the following criteria: (1) prospective design; (2) the exposure of interest was consumption of total dietary fiber, including fiber from cereal, fruit, vegetable, and other foods; (3) the endpoint of interest was fatal or nonfatal stroke; and (4) the relative risk (RR) or hazard ratio (HR), and the corresponding 95 % confidence interval (CI) were reported. Three authors (Z.Z., X.G., and L.X.) independently assessed the retrieved studies according to the selection criteria, and discrepancies were resolved by consensus.

Data extraction and quality assessment

For each study, the following characteristics were collected using a standardized data collection form: the first author's last name, publication year, study location, study period, length of follow-up, number of stroke cases and participants, range of dietary fiber consumption, age, sex, fiber consumption assessment method, outcome assessment method, RR from the most fully adjusted model for the highest compared with the lowest dietary fiber consumption and the corresponding 95 % CI, and adjustment for potential confounders in a multivariate analysis. Data were extracted independently by two authors (Z.Z. and G.X.) with disagreements resolved by consensus.

A 9-star system based on the Newcastle–Ottawa Scale [21] was adopted to evaluate the study quality. The included studies were assessed on 3 aspects: the selection of cohorts, comparability of cohorts, and ascertainment of outcome. The full score was 9 stars, and the high-quality study was defined as a study with $\geq$8 awarded stars. Study quality was also evaluated by 2 independent reviewers (Z.Z. and G.X.).

Statistical analyses

Relative risks or hazard ratios were extracted from the selected publications, and their SEs were calculated from the respective CIs. Heterogeneity among studies was assessed with the Q and I^2 statistic [22]. The combined risk estimates were calculated using either fixed-effects models or, in the presence of heterogeneity, random-effects models [23]. Subgroup analyses according to geographic region, length of follow-up, gender, stroke subtype, fiber source and fiber type, and quality score were performed to assess the potential effect modification of these variables on outcomes. Cumulative meta-analysis was also performed to demonstrate the trend in risk effect.

Because characteristics of cohorts, assessment methods of fiber consumption and endpoint, and adjustments for confounding factors were different among studies, we further carried out a sensitivity analysis to explore possible explanations for heterogeneity and to examine the influence of various exclusion criteria on the overall result. We also assessed the impact of a single study on the overall result by omitting each study in turn.

In the dose–response meta-analysis, we used the method proposed by Greenland and Longnecker [24] and Orsini et al. [25] to calculate the trend from the correlated log RR estimates across categories of fiber consumption. For every study, the median or mean fiber consumption for each category was assigned to each corresponding RR. We examined a potential nonlinear relationship between fiber consumption and stroke risk by modeling fiber consumption using restricted cubic splines with 3 knots at percentiles 25, 50, and 75 % of the distribution [26]. A P value for nonlinearity was calculated by testing the null hypothesis that the coefficient of the second spline is equal to 0.

Publication bias was assessed by funnel plots and Egger's linear regression test [27]. All analyses were performed using STATA version 12.0 (StataCorp, College Station, TX, USA). $P < 0.05$ was considered statistically significant.

Results

Literature search

A flow diagram of the literature search is shown in Fig. 1. Total searches yielded 504 entries. Of these, 488 articles were excluded after reading the title or abstract because of obvious irrelevance to our study aim. Sixteen full-text articles were reviewed [8–18, 28–32]. Three studies were excluded because they assessed the association between

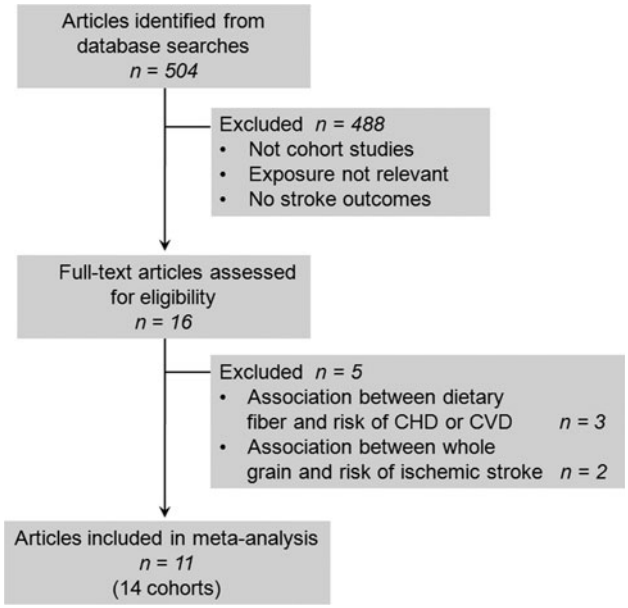

Fig. 1 Flowchart of study selection

dietary fiber consumption and risk of coronary heart disease [28] or total cardiovascular disease [29, 30]. We further excluded two studies that evaluated the association between whole grain consumption and risk of ischemic stroke [31, 32]. Therefore, a total of 11 studies containing 14 independent cohorts were included in our meta-analysis.

Study characteristics and quality assessment

The characteristics of the 11 studies are presented in Table 1. These studies were published between 1987 and 2013. The length of follow-up ranged from 8 to 18 years, with a median of 12 years. The sizes of the cohorts ranged from 859 to 86,387 (total 325,627). Dietary fiber consumption was assessed by food frequency questionnaire in 10 studies and 24-h dietary recall in 1 study. Case ascertainments were not consistent across studies, with most using medical record and death certificates. Most studies provided RRs that were adjusted for age (all 11 studies), smoking (10 studies), alcohol consumption (9 studies),

Table 1 Characteristics of the included studies

Study	Years	Country	Age (year)	Sex	Study period	No. of cohort	Follow up (year)	Fiber intake assessment	Outcome	Outcome assessment
Khaw et al. [8]	1987	USA	50–79	M/F	1972–1985	859	12	24-h dietary recall	Fatal stroke	Death certificates
Ascherio et al. [9]	1998	USA	40–75	M	1986–1994	43,738	8	FFQ	Fatal/nonfatal stroke	Medical records or autopsy reports
Mozaffarian et al. [10]	2003	USA	≥65	M/F	1989–2000	3,588	8.6	FFQ	Fatal/nonfatal stroke	Annual examinations, telephone interviews, medical records, physician questionnaires, death certificates, medical examiner forms
Oh et al. [11]	2005	USA	30–55	F	1980–1998	78,779	18	FFQ	Fatal/nonfatal stroke	Medical records, death certificates
Weng et al. [12]	2008	China	≥40	M/F	1989–2002	1,772	10.6	FFQ	Fatal/nonfatal stroke	Death certificates, insurance claim records of NHI database, subject's self-reported disease history
Larsson et al. [13]	2009	Finland	50–69	M	1985–2004	26,556	13.6	FFQ	Fatal/nonfatal stroke	Discharge diagnoses and death certificates (ICD-8, -9, -10)
Kaushik et al. [14]	2009	Australia	≥49	M/F	1992–2005	2,897	13	FFQ	Fatal stroke	National Death Index
Eshak et al. [15]	2010	Japan	40–79	M/F	1988–2003	58,730	14.3	FFQ	Fatal stroke	Death certificates (ICD-10)
Kokubo et al. [16]	2011	Japan	45–65	M/F	1995–2004	86,387	10.4	FFQ	Fatal/nonfatal stroke	Medical records, death certificates (ICD-10)

Table 1 continued

Study	Years	Country	Age (year)	Sex	Study period	No. of cohort	Follow up (year)	Fiber intake assessment	Outcome	Outcome assessment
Wallstrom et al. [17]	2012	Sweden	44–73	M/F	1991–2006	20,674	13.5	FFQ, diet history, diet interview	Fatal/nonfatal stroke	Medical record from national patient register, national cause of death register, and stroke register
Casiglia et al. [18]	2013	Italy	≥18	M/F	1989–2000	1,647	12	FFQ	Fatal/nonfatal stroke	Register's office, hospitals, retirement homes or physicians' files, physicians and retirement homes records

Study	Sex	Participant	Stroke case	Comparison, g/day	Fiber intake difference, g/day	Adjustment for Covariates
Khaw et al. [8]	M/F	859	24	Continuous variable	6.0	Age, sex, potassium and calories
Ascherio et al. [9]	M	43,738	328	Q1: 12.4 Q5: 28.9	16.5	Age, total energy, smoking, alcohol consumption, history of hypertension and hypercholesterolemia, parental history of MI, profession, BMI, physical activity, and magnesium
Mozaffarian et al. [10]	M/F	3,588	344	Q1: 0.8 Q5: 7.9	7.1	Age, sex, education, diabetes, smoking, daily physical activity, exercise intensity, and alcohol intake
Oh et al. [11]	F	78,779	1,020	Q1: 10.0 Q5: 21.0	11.0	Age, BMI, smoking, alcohol intake, parental history of MI, history of hypertension, hypercholesterolemia and diabetes, menopausal status and postmenopausal hormone use, aspirin use, multivitamin use, vitamin E supplement use, physical activity, energy, and carbohydrate intake
Weng et al. [12]	M/F	1772	132	Q1: <24.586 Q3 + 4: >29.68	5.1	Age, sex, age*sex, hypertension, use of antihypertensive drugs, diabetes mellitus, area, central obesity, BMI, alcohol, smoking, sex*smoking, self-report heart disease, hypercholesterolemia, hypertriglyceridemia, physical activity, fibrinogen, apolipoprotein B, and plasminogen
Larsson et al. [13]	M	26,556	3,365	Q1: 16.1 Q5: 35.8	19.7	Age, smoking, BMI, blood pressure, total serum cholesterol, serum high-density lipoprotein cholesterol, histories of diabetes and coronary heart disease, leisure-time physical activity, intakes of alcohol and total energy, and intakes of folate and magnesium
Kaushik et al. [14]	M/F	2897	95	T1: 3.0 T3: 11.0	8.0	Age, sex, blood pressure, antihypertensive medication use, BMI, smoking, educational qualifications, fair or poor self-rated health, history of MI and stroke, and presence of diabetes

Table 1 continued

Study	Sex	Participant	Stroke case	Comparison, g/day	Fiber intake difference, g/day	Adjustment for Covariates
Eshak et al. [15]	M	23,119	499	Q1: <7.8 Q5: >12.6	6.4	Age, BMI, history of hypertension, history of diabetes, alcohol consumption, smoking, education, hours of exercise, hours of walking, perceived mental stress, sleep duration, fish, SFA, (n-3) fatty acids, sodium, folate, and vitamin E
	F	35,611	484	Q1: <8.5 Q5: >12.7	5.7	
Kokubo et al. [16]	M	40,046	1,499	Q1: 6.0 Q5: 19.9	13.9	Age, sex, smoking, alcohol, BMI, history of diabetes, medications for hypertension and hypercholesterolemia, exercise, dietary intakes of fruits, vegetables, fish, sodium, isoflavone and energy, and public health center
	F	46,341	1,054	Q1: 7.8 Q5: 21.6	13.8	
Wallstrom et al. [17]	M	8139	401	Q1: 5.8 Q5: 11.4	5.6	Age, diet assessment method version, total energy intake, season, BMI, smoking, education, alcohol category, systolic blood pressure, antihypertensive treatment, antihyperlipidemic treatment, and physical activity
	F	12,535	354	Q1: 6.5 Q5: 12.9	6.4	
Casiglia et al. [18]	M/F	1647	77	>25 vs ≤25 for soluble fiber >47 vs ≤47 for insoluble fiber	–	Age, gender, smoking, diabetes, arterial hypertension, hyperuricaemia, obesity, left ventricular hypertrophy, dyslipidaemia, chronic pulmonary disease, hyperuricaemia, dietary intake of lipids, carbohydrates, proteins, caffeine and ethanol intake, and vegetable/met ratio

FFQ food frequency questionnaire, *ICD* International classification of diseases, *MI* myocardial infarction, *Q* quintile, *SFA* saturated fatty acid, *T* tertile

history of hypertension or measured blood pressure (9 studies), body mass index (8 studies), physical activity or exercise (8 studies), history of diabetes (8 studies), and other nutrients (7 studies). Quality scores of each studies are summarized in Table 2. The range of quality scores was from 5 to 9, and the median score was 8.

Fiber consumption and stroke risk

The multivariable-adjusted RRs for each study and the pooled RR for the highest versus the lowest categories of dietary fiber consumption are shown in Fig. 2. Among the 11 studies, 10 showed an inverse association between dietary fiber consumption and stroke risk, 5 of which [10, 14, 16–18] were statistically significant. An opposite trend was observed in only 1 cohort. Overall, the pooled RR of stroke for the highest compared with the lowest dietary fiber consumption was 0.83 (95 % CI 0.74, 0.93) with marginal heterogeneity among studies ($P = 0.07$, $I^2 = 38.7$ %). Starting with the first published study we calculated the cumulative pooled RR by stepwise addition of the results of the other available studies up to the last one published in 2013. In the cumulative meta-analysis, the pooled RR achieved significance starting in 2003 ($P = 0.006$) and showed a trend of association as published data accumulated ($P = 0.001$, Fig. 3).

Table 2 Methodologic quality of cohort studies included in the meta-analysis

Author/year	Representativeness of the exposed cohort	Selection of the non exposed cohort	Ascertainment of exposure	Outcome of interest not present at start of study	Control for important factors[a]	Assessment of outcome	Follow-up long enough for outcomes to occur[b]	Adequacy of follow up of cohorts[c]	Total quality scores
Khaw et al. [8].	☆	☆	–	☆	☆☆	☆	☆	☆	8
Ascherio et al. [9].	–	☆	☆	☆	☆☆	☆	☆	☆	8
Mozaffarian et al. [10].	☆	☆	☆	☆	☆☆	☆	☆	–	8
Oh et al. [11].	–	☆	☆	–	☆☆	☆	☆	☆	7
Weng et al. [12]	☆	☆	☆	☆	☆	☆	☆	☆	8
Larsson et al. [13].	–	☆	☆	☆	☆☆	☆	☆	–	7
Kaushik et al. [14].	–	☆	☆	–	☆	☆	☆	–	5
Eshak et al. [15].	–	☆	☆	☆	☆☆	☆	☆	–	7
Kokubo et al. [16].	–	☆	☆	–	☆☆	☆	☆	–	6
Wallstrom et al. [17].	☆	☆	☆	☆	☆☆	☆	☆	☆	9
Casiglia et al. [18]	☆	☆	☆	–	☆☆	☆	☆	☆	8

[a] A maximum of 2 stars could be awarded for this item. Studies that controlled for age received one star, whereas studies that controlled for intake of other nutrients received an additional star

[b] A cohort study with a follow-up time >5 years was assigned one star

[c] A cohort study with a follow-up rate >80 % was assigned one star

Stratifying analysis

Stratifying by geographic region, the RRs were 0.80 (95 % CI 0.70–0.92) for studies conducted in the United States, 0.76 (95 % CI 0.57–1.02) for studies in Europe, and 0.92 (95 % CI 0.72–1.17) for studies in Japan (Table 3). The association between dietary fiber consumption and risk of stroke was similar in studies with ≤12 years of follow-up (RR 0.79; 95 % CI 0.68–0.92) and in studies with >12 years of follow-up (RR 0.89; 95 % CI 0.80–0.99). Stratifying by gender, the combined RRs of stroke were 0.94 (95 % CI 0.83–1.08) in male and 0.80 (95 % CI 0.66–0.96) in female. In the subgroup analyses by stroke subtype, the pooled RR for the highest compared with the lowest dietary fiber consumption was 0.83 (95 % CI 0.74, 0.93) for ischemic stroke and 0.87 (95 % CI 0.72, 1.05) for hemorrhagic stroke (Fig. 4). In addition, in the subgroup analyses by fiber source and fiber type, the RRs were 0.76 (95 % CI 0.58–1.00) for cereal fiber, 0.92 (95 % CI 0.83–1.02) for fruit fiber, 0.86 (95 % CI 0.77–0.95) for vegetable fiber, 0.76 (95 % CI 0.57–1.03) for insoluble fiber, and 0.78 (95 % CI 0.61–1.01) for soluble fiber (Fig. 5). Moreover, significant association was observed in high quality studies (≥8 stars).

Dose–response meta-analysis

We next evaluated the dose–response relationship between dietary fiber consumption and risk of stroke. Six studies were not eligible for the dose–response analysis because they did not provide the required data [10–13, 18] (e.g., the number of cases in each category) or data were based on continuous variables [8]. We found no evidence of a non-linear relationship between dietary fiber consumption and risk of stroke (P for nonlinearity = 0.15). Notably, the increment in dietary fiber consumption was associated with decreased risk of stroke in a dose–response manner (RR 0.90, 95 % CI 0.82–0.99 for 5 g increment, 0.84, 0.75–0.94 for 10 g increment, and 0.77, 0.66–0.91 for 15 g increment; Fig. 6).

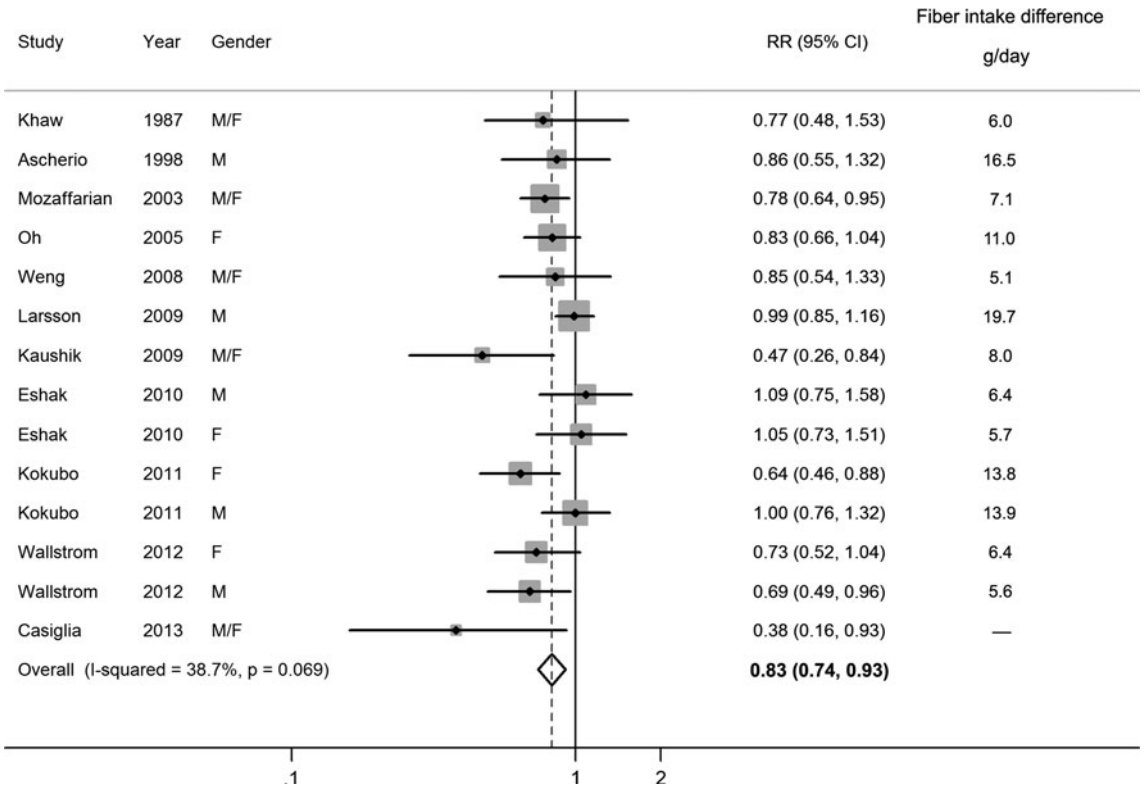

Fig. 2 Forest *plot* of prospective cohort studies examining dietary fiber consumption and risk of stroke. The *squares* and *horizontal lines* correspond to the study-specific RR and 95 % CI. The *diamond* represents the summary RR and 95 % CI

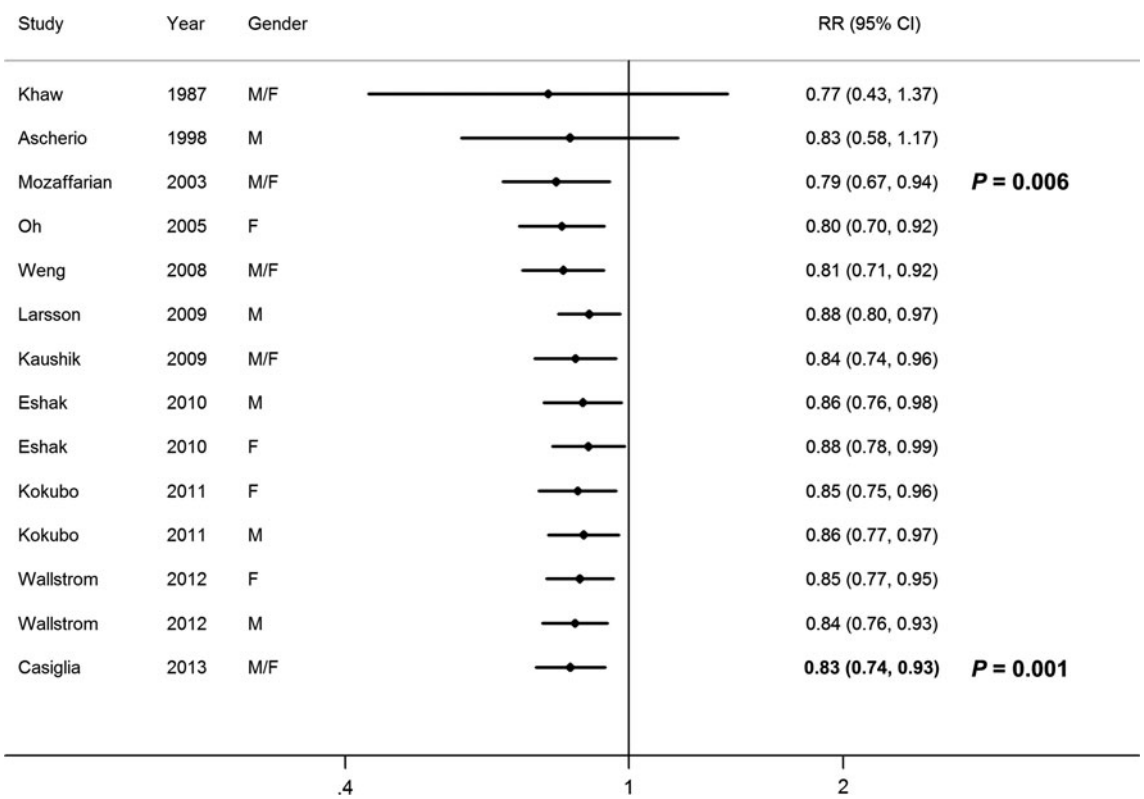

Fig. 3 Cumulative meta-analysis. Evaluation of time trends (year of publication) in relation between dietary fiber consumption and risk of stroke. The *squares* and *horizontal lines* correspond to the study-specific RR and 95 % CI

Table 3 Stratification analyses of dietary fiber consumption and stroke risk

Group	No. of studies	RR (95 % CI)	P-heterogeneity
Total	11	0.83 (0.74–0.93)	0.07
Geographic region			0.54
United States	4	0.80 (0.70–0.92)	
Europe	3	0.76 (0.57–1.02)	
Japan	2	0.92 (0.72–1.17)	
Length of follow-up			0.21
≤12 years	6	0.79 (0.68–0.92)	
>12 years	5	0.89 (0.80–0.99)	
Stroke subtype			0.68
Ischemic stroke	6	0.83 (0.74–0.93)	
Hemorrhagic stroke	4	0.87 (0.72–1.05)	
Gender			0.17
Male	5	0.94 (0.83–1.08)	
Female	4	0.80 (0.66–0.96)	
Stroke subtype and gender			0.26
Ischemic stroke in male	3	0.90 (0.72–1.12)	
Hemorrhagic stroke in male	2	0.98 (0.74–1.30)	
Ischemic stroke in female	3	0.74 (0.62–0.90)	
Hemorrhagic stroke in female	2	0.72 (0.52–0.98)	
Fiber source			0.37
Cereal fiber	4	0.76 (0.58–1.00)	
Fruit fiber	2	0.92 (0.83–1.02)	
Vegetable fiber	2	0.86 (0.77–0.95)	
Fiber type			0.90
Insoluble fiber	4	0.76 (0.57–1.03)	
Soluble fiber	4	0.78 (0.61–1.01)	
Quality score			
<8 stars	5	0.88 (0.75–1.04)	0.17
≥8 stars	6	0.76 (0.66–0.86)	

Sensitivity analyses

To test the robustness of our results, we conducted sensitivity analyses. Hypertension and diabetes were potential confounders of the association between dietary fiber consumption and stroke risk. The results persisted when we excluded the study [8] that did not adjust for hypertension or diabetes (RR 0.83, 95 % CI 0.74–0.93), with marginal heterogeneity ($P = 0.05$, $I^2 = 43.1$ %). Exclusion of 3 studies [8, 14, 15] in which fatal stroke served as endpoint changed the overall risk estimate little (RR 0.82, 95 % CI 0.73–0.92), without substantial heterogeneity ($P = 0.12$, $I^2 = 36.4$ %). Further exclusion of any single study did not materially change the overall combined RR, with a range from 0.80 (95 % CI 0.72–0.90) to 0.85 (95 % CI 0.76–0.95).

Fiber consumption and stroke mortality

Of the 11 included studies, three [8, 14, 15] reported a risk estimate of dietary fiber consumption with respect to stroke mortality. In the pooled analysis, there was a trend toward an inverse association between higher fiber consumption and risk of stroke mortality (RR 0.85; 95 % CI 0.60–1.20), which was not statistically significant ($P = 0.36$).

Publication bias

The funnel plot did not show substantial asymmetry. Egger's test also indicated no evidence of publication bias ($P > 0.05$).

Comment

The present meta-analysis involving 325,627 participants confirmed a significant inverse association between dietary fiber consumption and stroke risk. The risk of stroke was reduced by 17 % in a comparison of the highest with the lowest categories of dietary fiber consumption. Moreover, the increment in dietary fiber consumption was associated with decreased risk of stroke in a dose–response manner.

There are several potential mechanisms by which dietary fiber may reduce risk of stroke. Hypertension is an important risk factor for stroke. The protective effect of dietary fiber against risk of stroke may in part be related to its blood pressure-lowering effect.

Meta-analyses of randomized placebo-controlled trials indicate that fiber supplementation (average dose, 11.5 g/day) changed systolic blood pressure by −1.13 mm Hg and diastolic blood pressure by −1.26 mm Hg [4]. In addition, fiber consumption has been shown to affect serum lipid levels, and insulin sensitivity [5, 6]. Moreover, fiber consumption could benefit endothelial cell function and prevent impaired vascular reactivity in response to a high fat meal [33]. Dietary fiber may also reduce stroke risk via a substitution effect, replacing consumption of other potentially detrimental foods.

In our study, dietary fiber consumption was associated with significantly reduced risk of ischemic stroke but not with hemorrhagic stroke. The nonsignificant association in hemorrhagic stroke was likely due to the small number of cases, which led to a lower statistical power to estimate this association. In addition, the inverse association between dietary fiber consumption and risk of stroke was more pronounced among females than males. The possible explanation is that compared with females, males are more likely to

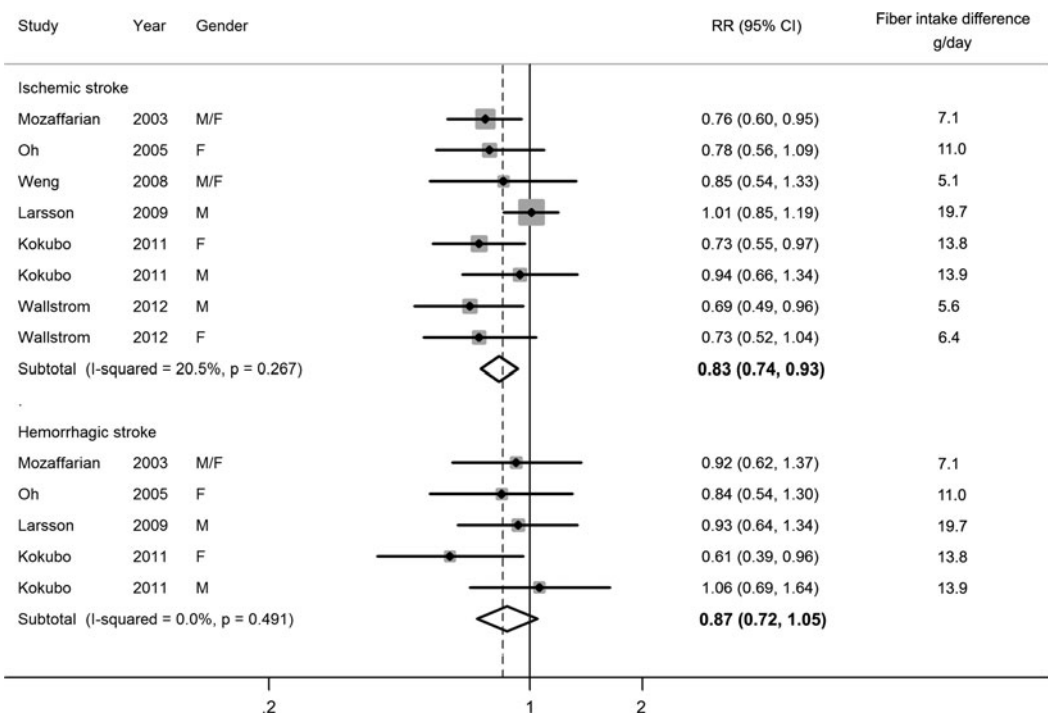

Fig. 4 Forest *plot* of prospective cohort studies examining dietary fiber consumption and risk of stroke stratified by subtype. The *squares* and *horizontal lines* correspond to the study-specific RR and 95 % CI. The *diamond* represents the summary RR and 95 % CI

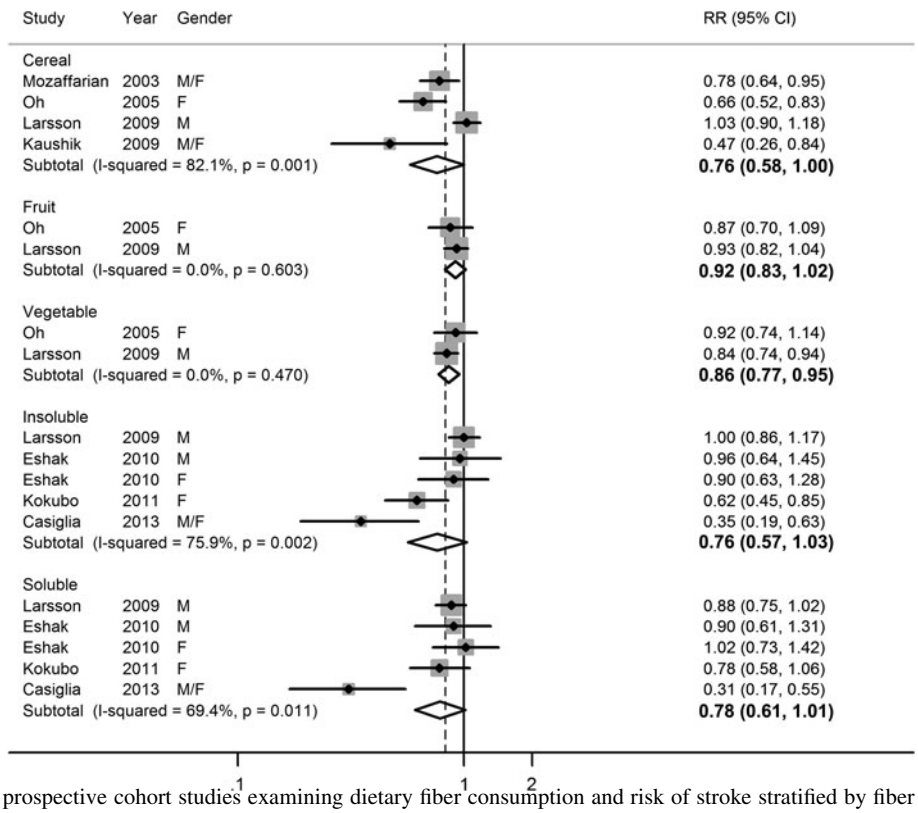

Fig. 5 Forest *plot* of prospective cohort studies examining dietary fiber consumption and risk of stroke stratified by fiber source and fiber type. The *squares* and *horizontal lines* correspond to the study-specific RR and 95 % CI. The *diamond* represents the summary RR and 95 % CI

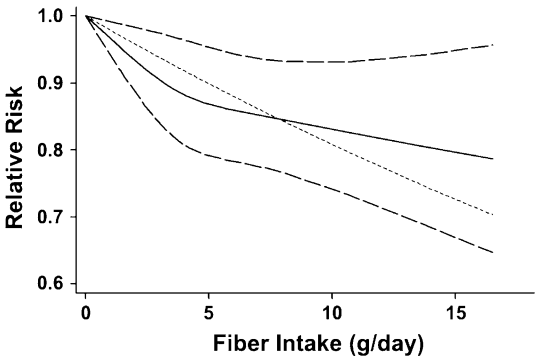

Fig. 6 Relative risk (*solid line*) with 95 % CI (*long dashed lines*) for the association of dietary fiber consumption with risk of stroke in a restricted cubic spline random-effects meta-analysis. The *short dash line* represents the linear trend. The lowest value of 3 g/day of dietary fiber consumption was used for the estimation of all relative risks

be smokers and drinkers, and consume more calories, which could diminish the benefit of dietary fiber.

Moreover, 4 studies [13, 15, 16, 18] examined the association of different types of dietary fiber (soluble or insoluble) with stroke risk. Although we observed inverse associations for both types of fiber, the association were stronger for soluble fiber. A characteristic of soluble fiber that may explain this result is its propensity to increase intraluminal viscosity of the small intestine, thus slowing the absorption of nutrients and potentially binding bile acids [34]. These effects could improve glucose control [35], lower serum cholesterol levels [36], and possibly lower blood pressure [37]. With regard to fiber source, higher cereal fiber and vegetable fiber but not fruit fiber was associated with decreased stroke risk, indicating that different sources of fiber may have varying effects in stroke prevention. Because of the limited available studies regarding fiber types and fiber sources included in the meta-analysis, the results should be interpreted with caution. Future studies concerning these factors are needed.

Our results confirmed the findings of previous meta-analysis [19]. With the accumulative evidence, we can enhance the precision of the risk estimates and carry out stratification analyses to explore possible sources of heterogeneity, thus increasing the clinical relevance of our results. Second, most prospective cohort studies in our meta-analysis had a large number of participants and stroke cases, and had a long period of follow-up, which significantly increased the statistical power of our study to assess the long-term effects of dietary fiber consumption on stroke risk. Third, our meta-analysis was based on prospective studies, which will minimize the possibility of selection bias and recall bias that could be of concern in retrospective case–control studies. Fourth, we observed an obvious dose–response relation between dietary fiber consumption and risk of stroke, which further enhanced this association.

Limitations

Several limitations of this meta-analysis should be acknowledged. First, though most included studies adjusted for known risk factors for stroke, residual confounding cannot be excluded because of the observational design. Dietary fiber consumption tends to be associated with healthy lifestyles that may protect against stroke, such as less smoking and drinking, more physical activity, and higher folate intake. Second, there is a wide range of values for the cutoff points for the highest and lowest categories for dietary fiber consumption in different studies, which might also affect the current analysis. Thus, large randomized clinical trials should be conducted with uniform criteria for dietary fiber consumption in different countries.

Implications

Stroke is a leading cause of death and long-term disability worldwide. The association between dietary fiber consumption and risk of stroke remain conflicting rather than conclusive. Based on the findings of our meta-analysis, a 15-g/day increment in fiber consumption was associated with a reduction in the risk of stroke of 23 %. This risk reduction would be translated into a reduction of as many as 1,311,000 stroke deaths per year worldwide [38] and is expected to produce overall health benefits by reducing the influence of physical disabilities. The recommended amount of dietary fiber consumption is 20–35 g/day [7], while the average fiber consumption of the populations included in this meta-analysis was far lower than this. With regard to public health, increased consumption of fiber-rich foods, such as fruit and vegetable, may bring great benefits in stroke prevention, especially in those at high risk.

Conclusions

The results from this meta-analysis of prospective studies indicate that dietary fiber consumption is inversely associated with risk of stroke. Further large studies are needed to confirm our findings.

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Appendix

PRISMA 2009 Checklist

Section/topic	#	Checklist item	Reported on page #
TITLE			
Title	1	Identify the report as a systematic review, meta-analysis, or both.	1
ABSTRACT			
Structured summary	2	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	2
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known.	3
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	3-4
METHODS			
Protocol and registration	5	Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.	4
Eligibility criteria	6	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	4
Information sources	7	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	4
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	4
Study selection	9	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	4
Data collection process	10	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	4-5
Data items	11	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	4-5
Risk of bias in individual studies	12	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	5-6
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	5
Synthesis of results	14	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I^2) for each meta-analysis.	5
Risk of bias across studies	15	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	5-6
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	5-6
RESULTS			
Study selection	17	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	6-7
Study characteristics	18	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	7
Risk of bias within studies	19	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	7-8
Results of individual studies	20	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	7
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	7-8
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	9-10
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	8-9
DISCUSSION			
Summary of evidence	24	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	10
Limitations	25	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	12
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	13
FUNDING			
Funding	27	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	14

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