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Meta-analysis

Coffee and tea consumption and the risk for subarachnoid hemorrhage: A meta-analysis

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ABSTRACT

Objectives: Reports on the association between coffee or tea consumption and subarachnoid hemorrhage (SAH) risk are inconsistent. The aim of this study was to determine if an association exists between consumption of coffee or tea and the risk for SAH.

Methods: A random-effects model was used to estimate the summary relative risks (RRs) and 95% confidence intervals (CIs). Heterogeneity among studies was assessed using the statistics Cochran's Q and I^2. Seven studies on coffee consumption and five on tea consumption were included in the meta-analysis.

Results: The pooled RRs of SAH for the highest versus the lowest categories of coffee and tea consumption were 1.31 (95% CI, 0.84–2.05) and 0.83 (95% CI, 0.65–1.08), respectively. There was evidence of heterogeneity among studies of coffee consumption ($P_{heterogeneity} = 0.002$, $I^2 = 71.7\%$) but not among studies of tea consumption ($P_{heterogeneity} = 0.34$, $I^2 = 11.3\%$). Omitting one study that substantially contributed to the heterogeneity among studies of coffee consumption yielded a pooled RR of 1.51 (95% CI, 1.10–2.06). Dose-response analysis showed that the summary RRs of SAH for an increase of one cup of coffee and tea consumption per day were 1.00 (95% CI, 0.96–1.04) and 0.97 (95% CI, 0.85–1.11), respectively. There was no evidence of publication bias.

Conclusion: Our meta-analysis of current evidence does not support an association between the consumption of coffee or tea and SAH risk. Further studies with prospective designs that control for important confounders and provide sufficient data for dose-response analysis are warranted.

Introduction

Subarachnoid hemorrhage (SAH) is a subversive type of stroke that has a mortality rate of almost 50% [1,2]. Approximately 10% of patients die during the prehospital period, and survivors often suffer long-term neurologic or cognitive impairments due to the original hemorrhage and rehemorrhage, despite the development of novel treatment strategies [3–5]. Therefore, clarifying the risk factors of SAH remains important. Except for the most common risk factors of SAH, including hypertension, smoking, and heavy alcohol intake, the relation between diet and SAH has been a recent concern [6].

Coffee and tea—among the most widely used psychoactive substances in the world—are now the main sources of dietary caffeine. Caffeine has well-known, short-term cardiovascular effects, including increased plasma renin levels, peripheral vasoconstriction, increased blood pressure, and cardiac arrhythmias [7,8]. It was reported that coffee consumption could plausibly influence the risk for cardiovascular disease (CVD) due to its antioxidant properties [9]. Similarly, tea contains large quantities of polyphenols, which have various physiological effects, including antihypertensive and antithrombogenic effects, as well as low-density lipoprotein cholesterol oxidation prevention in vitro and in vivo [10]

To date, many epidemiologic studies examining the association between coffee and tea consumption and SAH risk have been published [11–19]. However, the results are inconsistent, with both positive and negative associations reported. This discrepancy may be due to different subject characteristics or study methodologies. Moreover, no quantitative summary of the evidence has ever been reported.

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Therefore, we conducted a meta-analysis of published observational studies to quantify whether coffee or tea consumption influences the risk for SAH.

Materials and methods

Literature search

Independent computerized literature searches were conducted on SAH in relation to coffee or tea consumption in the PubMed and Embase databases through February 2018 by two investigators. "Coffee," "caffeine," "beverages," "drink," "tea," "green tea," "black tea," and "subarachnoid hemorrhage" were searched as keywords and expanded as MeSH where possible. Furthermore, we reviewed citations in the retrieved articles to search for additional relevant studies. The present study was performed in accordance with the guidelines proposed by the MOOSE group [20].

Study selection

Studies were considered eligible if they met the following criteria:

1. The study was an original epidemiologic cohort or case-control study.
2. The exposure of interest was coffee or tea consumption and SAH risk.
3. The relative risk (RR) estimates and associated 95% confidence intervals (CIs) for the highest versus the lowest categories of coffee or tea consumption were reported.

If the same population was evaluated in more than one study, we included the study with the longest follow-up period.

Data extraction and assessment of study quality

Data from the included studies were extracted and summarized by two independent investigators via a standardized data extraction form. Information was recorded as follows: last name of the first author, publication data, duration of follow-up (for cohort studies) or period of enrollment (for case–control studies), study design, geographical location, sex and age of participants, number of cases and participants, risk estimates with the most fully adjusted model for the highest versus the lowest category of coffee or tea consumption and the corresponding 95% CI, and covariates selected in multivariable models.

Study quality was evaluated separately by two observers using the Newcastle-Ottawa Scale (NOS) criteria. Briefly, the NOS score is based on three aspects: selection, comparability and exposure (for case–control studies), or outcome (for cohort studies). In the current meta-analysis, we considered studies with more than six stars as being high quality. Any discrepancies were resolved by reevaluating the included articles and discussing the discrepancies with a third investigator.

Statistical analysis

RRs with 95% CIs were used to measure the association between coffee or tea consumption and SAH risk. As the prevalence of SAH was relatively low, odds and hazard ratios were directly considered to be RRs. As a conservative approach, we calculated the summary estimates using a random-effects model that accounted for between-study variations. Statistical heterogeneity among studies was assessed using the statistics Cochran's Q and I^2. Heterogeneity among studies was considered significant for P-values of Cochran's Q <0.10 and $I^2 >50\%$. When significant heterogeneity was observed, subgroup analyses were conducted according to the study design (cohort and case–control studies), location (Europe, Asia, and other countries), and study quality score (high: NOS >6; low: NOS ≤ 6). We also conducted a sensitivity analysis after removing one study, and the remaining studies were analyzed to evaluate whether the results changed significantly. Next, a dose–response analysis of coffee or tea consumption with SAH risk was performed based on the method proposed by Orsini et al [21]. We assessed a potential curvilinear association between coffee or tea consumption and SAH risk using restricted cubic splines with 3 knots at the 25%, 50%, and 75% percentiles of the distribution. A P-value for non-linearity was calculated by testing whether the coefficient of the second spline transformation was equal to zero against the null hypothesis. Only the studies that reported the number of cases, person-years or controls, and adjusted RRs with 95% CIs for at least three quantitative exposure categories were included. The average dose was assigned as the mean of the upper and lower bounds in each category. If the upper bound was not reported in individual studies for the highest category, the average intake in this category was set to 1.5 times the lower boundary.

We used Begg's funnel plots [22] and Egger's regression test to examine potential publication bias [23]. All statistical analyses were performed using STATA

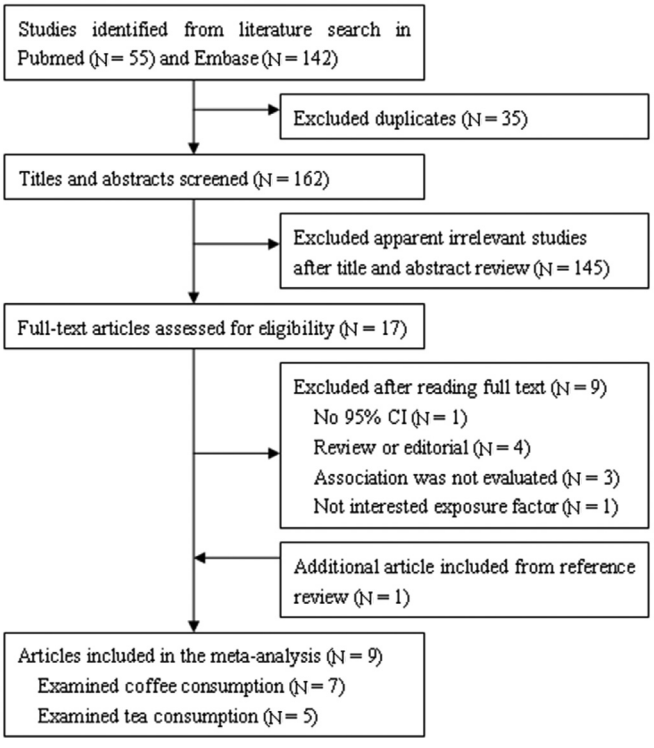

Fig. 1. Flowchart of study selection.

Table 1
Characteristics of the included studies

Author, year of publication (study period, y)	Design	Location	Sex	Age (y)	Cases/ participants (n)	Exposure variables	Contrast (highest vs lowest)	Adjustments	Quality score
Isaksen et al., 2002 (1986–1997)	Case–control study	Norway	F/M	NA	26/27 161	Coffee	>5 vs ≤5 cups/d	NA	7
Okamoto et al., 2006 (1992–1997)	Case–control study	Japan	F/M	30–79	201/NA	Tea	≥1 vs 0 × /d	History of hypertension, smoking, family history of SAH, education	5
Larsson et al., 2008 (1985–2004, 13.6)	Cohort study	Finland	M	50–69	196/26 556	Coffee and tea	≥8 vs <2 cups/d, ≥2 vs 0 cups/d	Age, supplementation group, smoking, BMI, alcohol intake, tea consumption	7
Jimenez-Yepes et al., 2008 (2004–2005)	Case–control study	Colombia	F/M	51	163/479	Coffee	NA	NA	7
Larsson et al., 2011 (1987–1990, 10.4)	Cohort study	Sweden	F	49–83	79/34 670	Coffee	≥5 vs <1 cup/d	Age, smoking, education, BMI, physical activity, history of diabetes, history of hypertension, aspirin use, family history of myocardial infarction, and intakes of total energy, alcohol intake	7
Shiue et al., 2011 (1995–1998)	Case–control study	Australia	F/M	16–94	383/856	Coffee and tea	>4 vs 1–4 × /wk, >4 vs 1–4 × /wk	Age, sex, ethnicity, source of information, education, BMI, history of hypertension, currently smoking, regular exercise, and all food items, including alcohol	6
Vlak et al., 2011	Case–control study	Netherlands	F/M	≥18	250/NA	Coffee and tea	≥1 vs 0 cups/d, ≥1 vs 0 cups/d	NA	5
Larsson et al., 2013 (1997–2008, 10.2)	Cohort study	Sweden	F/M	45–83	148/74,961	Tea	>2 vs 0 cups/d	Age, sex, smoking, BMI, physical activity, aspirin use, history of hypertension, diabetes, family history of myocardial infarction, and intakes of total energy, alcohol, and coffee	8
Sakamaki et al., 2016 (1992–1995, 10.7)	Cohort study	Japan	F/M	55	47/9941	Coffee	≥5 vs 0 cups/d	Age, sex, BMI, systolic blood pressure, total cholesterol concentration, smoking, and alcohol intake	9

BMI, body mass index; NA, not available; SAH, subarachnoid hemorrhage.

12.0 (Stata Corporation, College Station, TX, USA) statistical software. $P < 0.05$ was considered statistically significant unless otherwise specified.

Results

Literature search

We initially retrieved 197 unique citations from the PubMed and Embase databases. After removing duplicates and reviewing the titles or abstracts, 179 studies were considered ineligible. After reviewing the full texts of the remaining 18 articles, 9 were excluded for the following reasons: no 95% CIs (n = 1) were reported; the article was a review or an editorial (n = 4); associations were not evaluated (n = 3); an exposure factor of interest was not included (n = 1). One additional article was further included from the reference review [17]. Finally, nine observational studies [11–19] were eligible and included in the present meta-analysis. A flowchart summarizing the study search and selection procedure is presented in Figure 1.

Study characteristics

The general characteristics of the published articles included in this meta-analysis are shown in Table 1. The nine studies, including four cohort studies and five case–control studies, were all published in English between 2002 and 2016. Of the included studies, seven and five provided results for coffee and tea, respectively. Five studies were conducted in Europe, two in Japan, one in Columbia, and one in Australia. The sample sizes of the included studies ranged from 201 to 37 640 for a total of 174 924 samples, and the number of SAH cases varied from 26 to 383 for a total of 1493 SAH cases. Most risk estimates were adjusted for age, body mass index (BMI), family history of SAH, and smoking status. However, three studies [11,13,16] did not adjust for any cofounders. The NOS scores for the included studies varied from 5 to 9 points, with an average score of 6.8, indicating high quality.

Association between coffee consumption and SAH risk

Seven studies [11,13–17,19] presented results for the highest versus the lowest categories of coffee consumption and SAH risk. The results are shown in Figure 2. The pooled RR of SAH was 1.31 (95% CI, 0.84–2.05), with significant between-study heterogeneity ($P_{heterogeneity} = 0.002$, $I^2 = 71.7\%$).

Three studies [14,15,19] were included in the dose–response analysis of coffee consumption with SAH risk. Using a restricted cubic spline model, we found no evidence of a linear association between coffee consumption and SAH risk ($P = 0.49$ for non-linearity; Supplementary Fig. 1). The summary RR of SAH for an intake increase of one cup of coffee per day was 1.00 (95% CI, 0.96–1.04), with a low heterogeneity ($P_{heterogeneity} = 0.043$, $I^2 = 18.8\%$).

Association between tea consumption and SAH risk

The association between SAH risk and consumption of tea was reported in five studies [12,14,16–18]. Figure 3 shows a forest plot of the highest versus the lowest categories of tea consumption. The pooled RR was 0.83 (95% CI, 0.65–1.08). There was no statistically significant heterogeneity across these studies ($P_{heterogeneity} = 0.34$, $I^2 = 11.3\%$).

Two cohort studies [14,18] provided the data required for a dose-response analysis. We did not find a linear association between tea consumption and SAH risk ($P = 0.96$ for non-linearity; Supplementary Fig. 2). The combined RR of SAH for an increment of one

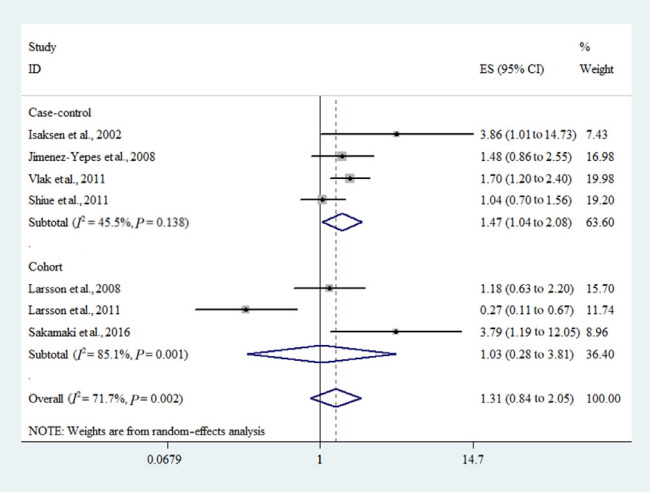

Fig. 2. Forest plot of coffee consumption and SAH risk. RR, relative risk; SAH, subarachnoid hemorrhage.

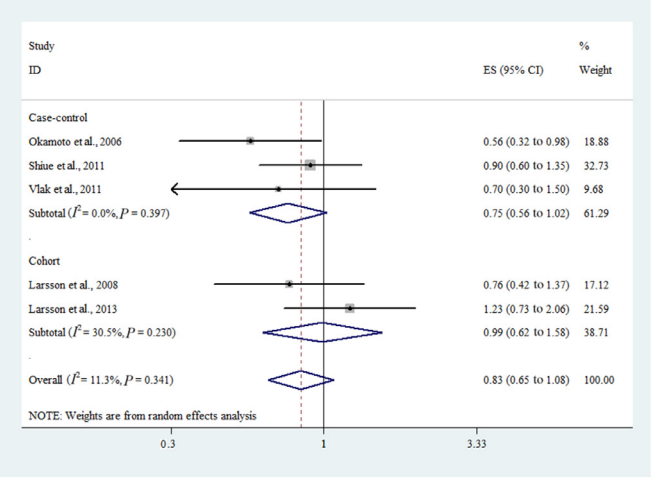

Fig. 3. Forest plot of tea consumption and SAH risk. RR, relative risk; SAH, subarachnoid hemorrhage.

cup per day of tea consumption was 0.97 (95% CI, 0.85–1.11) without heterogeneity ($P_{heterogeneity}$ = 0.42, I^2 = 4.96%).

Subgroup analysis

The results of subgroup analyses performed according to study design, geographical location, study quality score, and adjustments are presented in Table 2. Coffee consumption was associated with a significant increased risk for SAH among case–control studies (summary RR = 1.47; 95% CI, 1.04–2.08) but not among cohort studies (summary RR = 1.03; 95% CI, 0.28–3.81). When stratified by adjustments, a significantly positive association with SAH risk was observed among studies without control for confounders, such as age, gender, BMI smoking or alcohol intake, but not among studies adjusted for these confounders. Summary RRs stratified by geographical location and study quality score yielded results similar to those of the overall risk estimate for coffee consumption.

Similarly, the association between tea consumption and SAH risk was not significantly modified by study design or study quality score. However, when stratified by geographical location, a borderline significant inverse association was observed among Asian populations (summary RR = 0.56; 95% CI, 0.32–0.98) but not among European or other populations. Additionally, a significant inverse association was found when the analysis was restricted to studies without adjustments for age, sex, BMI, or alcohol intake but not studies with adjustments for these confounders.

Sensitivity analysis

In the sensitivity analysis, we excluded a single study to investigate the influence of the study on the overall risk estimate. For coffee consumption, the results showed that the study performed by Larrson et al. [15] substantially influenced the pooled RR. After excluding this single study, there was no significant between-study heterogeneity ($P_{heterogeneity}$ = 0.13, I^2 = 41.7%), and the RR for the highest versus the lowest category of coffee consumption was 1.51 (95% CI, 1.10–2.06) (Fig. 4). Regarding tea consumption, there were no changes in the direction of the effect when any one study was excluded, as shown in Figure 5.

We also performed a sensitivity analysis restricted to those studies that provided adjusted risk estimates. The combined RRs for coffee and tea consumption were 1.01 (95% CI, 0.48–2.14; $P_{heterogeneity}$ = 0.004, I^2 = 77.7%) and 0.85 (95% CI, 0.62–1.15; $P_{heterogeneity}$ = 0.23, I^2 = 30.2%), respectively.

Table 2
Subgroup and sensitivity analyses of association between coffee or tea consumption and subarachnoid hemorrhage risk

Group	Coffee				Tea			
	Studies (n)	RR (95% CI)	$P_{heterogeneity}$	I^2, %	Studies (n)	RR (95% CI)	$P_{heterogeneity}$	I^2, %
Total	7	1.31 (0.84–2.05)	0.002	71.7	5	0.83 (0.65–1.08)	0.341	11.3
Study design								
Case–control	4	1.47 (1.04–2.08)	0.138	45.5	3	0.75 (0.56–1.02)	0.397	0
Cohort	3	1.03 (0.28–3.81)	0.001	85.1	2	0.99 (0.62–1.58)	0.230	30.5
Geographic area								
Europe	4	1.15 (0.51–2.62)	0.001	81.7	3	0.93 (0.65–1.33)	0.360	2.1
Asia	1	3.79 (1.19–12.06)	–	–	1	0.56 (0.32–0.98)	–	–
Other countries	2	1.18 (0.85–1.64)	0.306	4.7	1	0.90 (0.60–1.35)	–	–
Study quality score								
High (NOS score >6)	5	1.36 (0.62–2.98)	0.001	77.4	2	0.99 (0.62–1.08)	0.230	30.5
Low (NOS score ≤6)	2	1.34 (0.83–2.17)	0.069	69.7	3	0.75 (0.56–1.02)	0.397	0
Adjusted for age								
Yes	4	1.01 (0.48–2.13)	0.004	77.7	3	0.95 (0.72–1.26)	0.457	0
No	3	1.70 (1.28–2.26)	0.430	0	2	0.60 (0.38–0.95)	0.655	0
Adjusted for sex								
Yes	4	1.01 (0.48–2.13)	0.004	77.7	2	1.01 (0.74–1.39)	0.352	0
No	3	1.70(1.28–2.26)	0.430	0	3	0.66(0.46–0.94)	0.752	0
Adjusted for BMI								
Yes	4	1.01 (0.48–2.13)	0.004	77.7	3	0.95 (0.72–1.26)	0.457	0
No	3	1.70 (1.28–2.26)	0.430	0	2	0.60 (0.38–0.95)	0.655	0
Adjusted for alcohol intake								
Yes	4	1.01 (0.48–2.13)	0.004	77.7	3	0.95 (0.72–1.26)	0.457	0
No	3	1.70 (1.28–2.26)	0.430	0	2	0.60 (0.38–0.95)	0.655	0
Adjusted for smoking								
Yes	4	1.01 (0.48–2.13)	0.004	77.7	4	0.85 (0.62–1.15)	0.231	30.2
No	3	1.70 (1.28–2.26)	0.430	0	1	0.70 (0.31–1.57)	–	–

BMI, body mass index; NOS, Newcastle-Ottawa Scale; RR, relative risk.

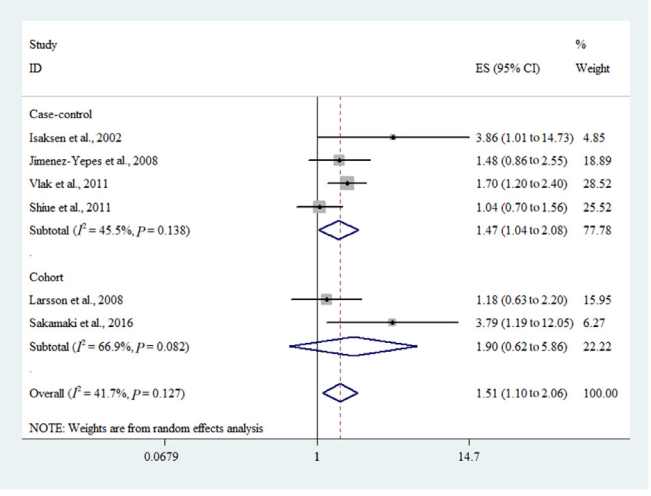

Fig. 4. Forest plot of coffee consumption and SAH risk after excluding the study performed by Larrson et al. [15]. RR, relative risk; SAH, subarachnoid hemorrhage.

Publication bias

The Begg's funnel plot does not show substantial asymmetry (Figs. 6, 7). Additionally, the Egger's regression test results show no evidence of publication bias (P = 0.98 for coffee consumption, P = 0.51 for tea consumption).

Discussion

The present meta-analysis summarized the results of observational studies, including seven studies [11,13–17,19] on coffee consumption and five studies [12,14,16–18] on tea consumption. Findings from the present study suggested that high coffee consumption is not associated with SAH risk. However, the sensitivity analysis results indicate a significantly increased risk for SAH after omitting one study [15] that substantially contributed to the heterogeneity among studies of coffee consumption. Additionally, there was no significant association between consumption of tea and SAH incidence.

The observed heterogeneity among studies of coffee consumption and SAH risk could be due to one large cohort study performed in Sweden [15]. The exclusion of this single study revealed a significant positive association between coffee consumption and SAH risk (51% increased risk when comparing the highest versus the lowest intake) without significant study heterogeneity. Compared with the other studies, the disparate results of the Swedish cohort may be due to the use of a self-administered questionnaire for assessing coffee consumption, which could inevitably lead to measurement

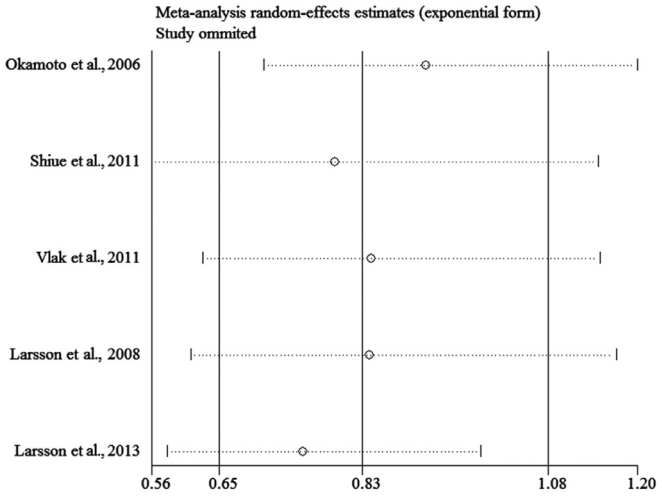

Fig. 5. Sensitivity analysis of tea consumption and SAH risk. SAH, subarachnoid hemorrhage.

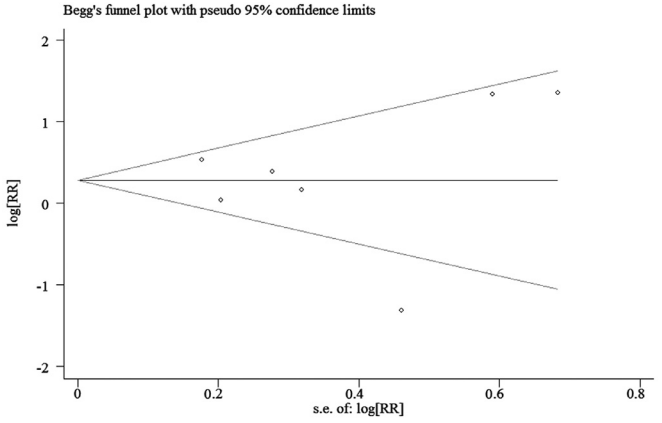

Fig. 6. Publication bias of coffee consumption and SAH risk. SAH, subarachnoid hemorrhage.

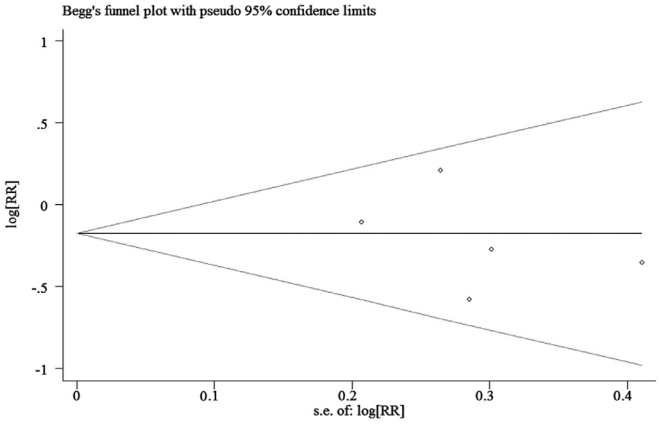

Fig. 7. Publication bias of tea consumption and SAH risk. SAH, subarachnoid hemorrhage.

error and misclassification of exposure. Alternatively, as the participants in that study were women 49 to 83 y of age, inadequate control for confounding factors, such as menopausal status and estrogen use, involved in caffeine metabolism and SAH incidence may have biased the results.

There are several speculated mechanisms by which high coffee consumption may increase the risk for SAH. One possible explanation is that excess consumption of caffeine, the main functional component in coffee, may increase systemic vascular resistance and then elevate blood pressure [24]. Another possible explanation is that hydroxyhydroquinone, which is generated by roasting coffee beans, could prevent the vasodilatory effect of chlorogenic acids [25], which exert antioxidant effects [26] that benefit vascular health [27,28]. Additionally, the intake of high-energy substances that often are added to coffee, such as sugar, milk, and cream, may induce oxidative stress and insulin resistance, which are related to

CVD [29–31]. Further research at the molecular level is required to elucidate the exact mechanism.

The present meta-analysis documented a positive association between coffee consumption and SAH risk in case-control studies, whereas no significant overall association was found in cohort studies. Of note, all case-control studies [11,13,16,17] included in the analysis reported exposure information obtained after the diagnosis of SAH; as such, these data may be subject to recall bias. Moreover, three [11,13,16] of the four included case-control studies did not adjust for any potential confounding factors, including smoking and alcohol intake, which may exaggerate the true association. Finally, selection bias is a concern in case–control studies. Thus, the overall finding of increase in SAH risk among case-control studies should not be overemphasized.

For tea consumption, the majority of studies reported a null association with SAH risk, except for a case-control study in Japan [12], which demonstrated that higher green tea consumption was borderline associated with a lower risk for SAH (odds ratio, 0.56; 95% CI, 0.32–0.98). Notably, the participants included in the study by Okamoto et al. were Asian, whereas the participants in all other studies were white. According to the level of fermentation, tea is divided into black tea (fermented), which is primarily consumed in Europe, North America, and North Africa, and green tea (unfermented), which is principally consumed in Asia [32–34]. It was previously reported that the protective effect of lowering stroke risk exerted by green tea tended to be greater than that of black tea [35]. This difference may be attributed to the disparate contents of polyphenolic compounds, especially catechins, in green and black tea [32]. Babu et al. showed that catechins induce a wide range of beneficial effects against CVD, including antihypertensive, antiendothelial dysfunction, and lipid-lowering effects [36]. Thus, although the present meta-analysis did not document a link between tea consumption and SAH risk, a possible beneficial effect of green tea against SAH incidence cannot be ruled out. However, due to the scarcity of data on the intake of different types of tea (i.e., green versus black tea), we did not perform a stratification analysis by tea subtype. Additional studies are needed to determine possible correlations.

Several limitations of this meta-analysis should be acknowledged when interpreting the results. First, our meta-analysis only contained nine studies, which may not be sufficient for drawing definitive conclusions about the correlation between coffee or tea consumption and SAH risk. Second, all included studies were observational designs, which are prone to various other sources of bias and confounding factors. For example, measurement bias could influence the findings, as assessments of coffee and tea intake largely depend on questionnaires. Third, as the dose-response analyses were based on a limited number of studies, the results should be interpreted with caution. Additionally, the average dose in each category was not reported in the included studies; therefore, the estimated value may not reflect the actual consumption of coffee and tea. Fourth, the adjusted covariates in individual studies differed, and three studies [11,13,16] did not control for any confounding factors. Although the sensitivity analysis restricted to studies reporting adjusted risk estimates revealed similar results, the lack of control for important confounding factors, such as age, sex, BMI, smoking and alcohol intake, could increase the risk for confounding bias, as suggested by our subgroup analysis. Fourth, because the relative composition of bioactive compounds in coffee varies by coffee preparation method, for example, boiled coffee contains much higher concentrations of diterpenes than filtered coffee [37], this could contribute to the heterogeneity among studies in different populations. However, the effect of different brewing methods on the risk for incident SAH has not been considered in the included studies. Therefore, we could not exclude the likelihood that the coffee brewing method may bias our findings. Finally, publication bias may have influenced the results. Although the Begg's and Egger's test results did not provide evidence of publication bias, the analyses were underpowered because of the small number of included studies.

Conclusion

Overall, the findings of this meta-analysis using observational studies did not support an association between the consumption of coffee or tea and SAH risk. Due to the limitations acknowledged, further studies with prospective designs that control for important confounders and provide sufficient data for dose-response analysis are warranted.

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Supplementary materials

Supplementary data related to this article can be found at https://doi.org/doi:10.1016/j.nut.2018.06.026.

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