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Tea Intake and Risk of Oral, Pharyngeal, and Laryngeal Carcinoma: A Meta-Analysis

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABDF 1 Wendong Zhang
CD 2 Tao Geng
BEG 1 Wenfei Han
ABCEF 3 Huiqin Dou

1 Department of Clinical Oncology, Qilu Hospital of Shandong University, Jinan, Shandong, China
2 Department of Pharmacy, Affiliated Hospital of Taishan Medical College, Tai'an, Shandong, China
3 Department of Stomatology, Qilu Hospital of Shandong University, Jinan, Shandong, China

Corresponding Author: Huiqin Dou, e-mail: douhuiqin76@126.com
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Background: The association between tea intake and risk of oral, pharyngeal, and laryngeal carcinoma is still unclear. The aim of this meta-analysis was to quantify the effect of tea consumption on the incidence of oral, pharyngeal, and laryngeal cancer to provide a better understanding on this issue.

Material/Methods: A literature search was conducted before January 2014 in MEDLINE and EMBASE databases. The relative risk (RR) estimates that extracted or calculated from all included studies were combined together. Given the existing heterogeneity in the study design and data source, a random-effects model was obtained.

Results: A total of 20 articles were included in the quantitative synthesis. Fourteen RR estimates (11 from case-control studies and 3 from cohort studies) were pooled together and the result demonstrated that tea consumption reduced the incidence of oral cancer (RR=0.85; 95% CI 0.76–0.96). The summary RR of 4 observational studies (3 case-control studies and 1 cohort study) for pharyngeal cancer was 0.87 (95% CI 0.74–1.04). The association between tea consumption and oral and pharyngeal carcinoma was reported. The summary RR for laryngeal carcinoma was 1.05 (95% CI 0.70–1.57). The Begg's funnel plot and the Egger's test showed no evidence of publication bias.

Conclusions: Tea consumption was associated with decreased risk of oral cancer, while no association was detected with oral/pharyngeal, pharyngeal, or laryngeal cancer.

MeSH Keywords: Administration, Oral • Meta-Analysis • Ocotea

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Background

Oral, pharyngeal, and laryngeal carcinomas are quite common world-wide. Recently, progress has been made through epidemiological studies investigating environmental risk factors for upper digestive tract cancer. Tobacco smoking and alcohol drinking are the major risk factors for these cancers [1]. It was reported that smoking and drinking are responsible of about 75% of cases diagnosed in North America and Europe [2]. The opinion that dietary factors may play an important role in the development of the oral, pharyngeal, and laryngeal cancer is not surprising. Considering that many ingested substances, including dietary carcinogens and anticancer substances, might affect in the incidence of cancers, a series of relevant epidemiological studies were conducted. Several diets included meat, vegetables, and fruits and the risk of these cancers were reported [3,4].

Tea is the most commonly consumed hot beverage in the world [5]. Tea consumption is reported to be associated with the development and progression of several kinds of chronic diseases [6]. In an *in vivo* study, preventive effects of green tea catechins were detected on spontaneous stroke in rats [7]. In an animal study, tea catechins dose-dependently reduced the brain infarct area and volume. Thus, daily intake of green tea may protect the penumbra from irreversible damage due to cerebral ischemia and consequent neurologic deficits [8]. Various experimental and clinical studies suggesting the role of green tea catechins against the markers of cardiovascular disorders and the underlying mechanisms for these actions have been discussed in a review [9]. A randomized clinical trial revealed that mildly hypertensive type 2 diabetic individuals who drink 3 glasses of green or sour tea daily for 4 weeks showed significantly decreased systolic and diastolic blood pressures [10]. Tea has long been regarded as a potential anti-cancer substance and is reported to decrease risk of several cancers [11,12]. The association between tea consumption and oral, pharynx, and larynx cancers were reported, but no clear conclusion was reached.

To the best of our knowledge, no systematic evaluation has been conducted on the association between tea consumption and oral, pharyngeal, and laryngeal carcinoma. Thus, we performed a systemic review and a meta-analysis to investigate the potential association. The aim of this meta-analysis was to quantify the effect of tea consumption on the risk of oral, pharyngeal, and laryngeal cancer to provide better understanding of this issue.

Material and Methods

Search strategy and inclusion criteria

We conducted this meta-analysis following the PRISMA guidelines [13] and MOOSE guidelines [14]. The literature search was conducted before January 2014 in MEDLINE and EMBASE databases. The following keywords were used in the literature search: (tea OR green tea OR black tea OR coffee OR caffeine OR beverages OR diet) and ((oral OR pharyngeal OR laryngeal) AND (cancer OR carcinoma)). Moreover, we searched for the additional relevant studies in the selected articles and published reviews. No language or any other restrictions were set in the search strategy. If necessary, we contacted the corresponding author of the article. No attempt was made to obtain unpublished data. Where the data sets were duplicated in different studies, only the most recent studies with sufficient data reported were included.

All identified studies were reviewed independently for eligibility by 2 authors. Citations selected from the initial search were subsequently screened for inclusion. Studies were included if they met the following criteria: 1) reported the association between tea consumption and incidence of oral, pharyngeal, or laryngeal carcinoma; 2) had a cohort or case-control design; and 3) indicated relative risk (RR) and odds ratios (OR) estimates with 95% CIs (or the raw data needed to calculate these).

Data extraction and assessment of study quality

The data of each included article was extracted by 2 reviewers independently and discussed to reach a consensus. The following variables were recorded: name of first author, publication year, study site, sex and age distribution of participants, sample size (number of cases and controls), exposure range, factors adjusted for in the analysis, and OR/RR estimates with corresponding 95% CIs for the highest versus lowest categories of tea drinking. We extracted the adjusted RR when possible, and unadjusted RR was calculated from the raw data.

The study quality was assessed by using the 9-stars Newcastle-Ottawa Scale (NOS) [15]. The 9-stars NOS assessed the selection, comparability, and exposure of a case-control study and the selection, comparability, and outcome of a cohort study. Studies with over 6 stars are considered to be relatively high quality.

Statistical methods for the meta-analysis

The RR estimates that extracted or calculated from all included studies were combined together. Given the existing heterogeneity in the study design and data source, a random-effects model was used in this study. If only stratified results (e.g., by cancer site) were provided in the primary studies, a fixed-effects method was obtained to summarize the outcomes into a single parameter for each study [16]. The effects of tea consumption on the incidence of oral, pharyngeal, or laryngeal carcinoma were measured with the RR with 95% CI.

For the meta-analysis, the heterogeneity of the associations across the included studies was assessed using the χ^2 test

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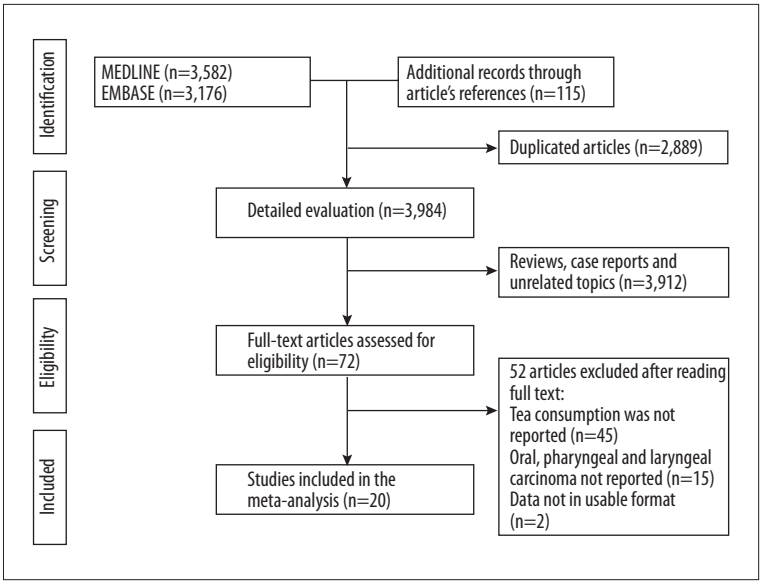

Figure 1. PRISMA diagram of included studies.

and I^2 score. The results were defined as heterogeneous for P<0.10 or I^2 >50%. Sensitivity analysis was performed to evaluate the robustness of the conclusion. Publication bias was visually examined by Begg's funnel plot [17] and statistically tested by Egger's regression asymmetry test [18]. All statistical tests were performed with the STATA software package (version 11.0; Stata Corporation, College Station, TX).

Results

Identification and selection of studies

A flow graph of our literature search is shown in Figure 1. The initial literature searches yielded 6873 entries (3582 from MEDLINE, 3176 from EMBASE, and 115 from the relevant reference lists). After the removal of 2889 duplicates, 3984 titles and abstracts were evaluated in detail. A total of 3912 articles were excluded because they were reviews, case reports, or studies with unrelated topics. Full texts of 114 articles were evaluated for inclusion. Fifty-two articles were excluded based on the following: tea consumption was not involved (n=45), oral, pharyngeal, or laryngeal carcinoma incidences were not reported (n=15), and data were not in usable format (n=2). The remaining 20 articles were included in the quantitative synthesis [19–38].

Study characteristics and quality

Among all the 20 included studies, the association between tea and the relevant cancers were reported: oral carcinoma in 13 articles, oral/pharynx carcinoma in 6 studies, pharynx carcinoma in 4 studies, and laryngeal carcinoma in 6 studies. When the incidence of oral cancer and pharyngeal cancer was pooled together in the primal articles, they were extracted and analyzed independently. A total of 11 984 cases were indemnified in this current meta-analysis. All the detailed characteristics of each included study are presented in Table 1. The included studies were published between 1987 and 2013. A total of 4 cohort studies and 16 case-control studies were identified in this current meta-analysis. Among all the included studies, 8 studies were in Europe, 6 in the Americas, 5 in Asia, and 1 in Africa. Of the 20 total included studies, 18 included the adjusted OR/RRs. The age, sex distribution, categories of tea consumption, and adjustments of the confounding factors are shown in Table 1.

The NOS were obtained to assess the selection, comparability, and exposure of the case-control studies and the selection, comparability, and outcomes of the cohort studies. Overall, most studies included in this meta-analysis had high quality (over 6 stars).

Tea consumption and risk of oral, pharyngeal, and laryngeal carcinoma

Figure 2 showed the RRs for the highest versus the lowest tea drinking level, as categorized in each study, for oral, pharyngeal, and laryngeal carcinoma. For oral cancer, 14 RR estimates (11 from case-control studies and 3 from cohort studies) were pooled together and the results demonstrated that tea consumption reduced the incidence of oral cancer (RR=0.85; 95% CI 0.76–0.96). In the stratifying analysis by study design, however, no significant association was detected in the case-control study (RR=0.87; 95% CI 0.75–1.01) or the cohort study (RR=0.80; 95% CI 0.61–1.05) (Figure 2A). The summary RR of 4 observational studies (3 case-control studies and 1 cohort study) for pharyngeal cancer (Figure 2B) was 0.87 (95% CI 0.74–1.04). Neither

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Table 1. Characters of the included studies.

Type	Author	Year	Study design	Age (year)	No. of case	Stratification	Adjusted factors
Oral	Notani PN, et al.	1987	Hospital/ Population CC Restropective	<40~>60	278	2, >2 cups/D *vs.* <2 cups/D	Age and tobacco habits
	Franceschi S, et al.	1992	Hospital prospective	<75	206	2, Intermediate, low	Age, area of residence, occupation, smoking, and alcohol habits
	Pintos J, et al.	1994	Hospital restropective	<54~>75	169	3, Never, ≤ 1,≥2 times/W	Tabcco, alcohol, income, rural residency, 10 dietary variables and consumption of other nonalcohol beverages
	Bundgaard T, et al.	1995	Population restropective	<45~>75	161	2, Nondrinker, drinker	Lifetime consumption of tobacco and alcohol
	Badawi AF, et al.	1998	Hospital restropective	47+15	42	2, <3, >3 cup/D	NA
	Galeone C, et al.	2010	Pooled data of 9 CC restropective	18–80	1130	3 Never, ≤1, >1 cups/D	Age, sex, race/ethnicity, edu-cation, study, cigarette smoking (pack-years), duration of cigar smoking, duration of pipe smoking, alcohol intake, weight, and vegetable and fruit intake
	Radoï L, et al.	2013	Population restropective	<50~>70	689	4 Never, <1, 1–2, >2 cups/D	Age, gender, area of residence, tobacco smoking (duration, quantity and status), alcohol consumption (quantity), education level, BMI two years before the interview and lifetime cumulative consumption of coffee
	Fu JY, et al.	2013	Hospital restropective	50~70	723	3 <4, 4–7, ≥8 cups/D	Age, region, education, smoking and alcohol drinking
	Franco EL, et al.	1989	Hospital restropective	<40~>70	232	3, <1, 1–3, >30 cup/M	Age, sex, study site, and admission period
	Zheng T, et al.	1993	Hospital restropective	18~80	404	2 Black, <1, >1 M; Green, <1, ≥1 M	Tobacco smoking, alcohol drinking, inadequate dentition, years of education, Quetelet Index, sex and age
	Zheng W, et al.	1996	Population prospective	55–69	71	4, Never/monthly, weekly, 1 cup/D, ≥2 cup/D	Age, education, smoking status, pack-years of smoking, physical acttvtty, a] fruit and vegetable Intake, walst/hlp ratio, and family history of cancer
	Ide R, et al.	2007	Population prospective	40–79	37	4, <1, 1–2, 3–4, >5 cup/D	Sex, age; smoking status; alcohol consumption; consumption of coffee, consumption of green/yellow vegetables, salty foods, and fruits
	Ren JS, et al.	2010	Population prospective	50–71	392	5, None, <1 cup/M, 1–3 cups/M 1–6 cups/W, ≥1 cup/d	Age, sex, tobacco smoking, alcohol drinking, BMI, education, ethnicity, usual physical activity throughout the D, vigorous physical activity and the daily intake of fruit, vegetables, red meat, white meat and calories
OP	Franceschi S, et al.	1999	Population restropective	22–77	598	1 2 3 4 5, cup/w	Age, centre, sex, education, smoking habit, total energy and alcohol intake
	Mashberg A, et al.	1993	Hospital restropective	37–80	359	3; 0, 1–2, >3 cup/D	Age, race, tobacco smoking, and alcohol drinking
	Tavani A, et al.	2003	Hospital CC restropective	<80	749	2, < 1, ≥1 cups/D	Terms for centre, age, sex, education, tobacco smoking, alcohol drinking, and intake of fruit and vegetables

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Table 1 continued. Characters of the included studies.

Type	Author	Year	Study design	Age (year)	No. of case	Stratification	Adjusted factors
OP	De Stefani E, et al.	2005	Hospital CC restropective	30–89	230	2, Nondrinker, drinker	Age, residence, urban/rural status, education, body mass index, smoking status, years since quitting, number of cigarettes smoked perD, alcohol drinking, and total energy intake
	La Vecchia C, et al.	1992	Population CC restropective	<85	119	2, Nondrinker, drinker	Age, sex, area of residence, education, smoking, and coffee consumption
	Hildebrand JS, et al.	2013	Population cohort prospective	>35	868	<1 cup/D 1–2 cups/D >2 cups/D	Age, sex, race, education, body mass index, alcohol use, smoking, vegetable intake, and intake of the other beverages
Pharynx	Notani PN, et al.	1987	Hospital/ population CC	<40~>60	225	2, >2 cups/D *vs.* <2 cups/D	Age and tobacco habits
	Pintos J, et al.	1994	Hospital CC restropective	<54~>75	112	3, Never ≤1, >1 cups/D	Tabcco, alcohol, income, rural residency, 10 dietary variables and consumption of other nonalcohol beverages
	Galeone C, et al.	2010	Pooled data of 9 CC restropective	18–80	2023	3 Never, ≤1, >1 cups/D	Age, sex, race/ethnicity, edu-cation, study, cigarette smoking (pack-years), duration of cigar smoking, duration of pipe smoking, alcohol intake, weight, and vegetable and fruit intake
	Ren JS, et al.	2010	Population cohort prospective	50–71	179	5, None, <1 cup/M, 1–3 cups/M 1–6 cups/W, ≥1 cup/d	Age, sex, tobacco smoking, alcohol drinking, BMI, education, ethnicity, usual physical activity throughout the D, vigorous physical activity and the daily intake of fruit, vegetables, red meat, white meat and calories
Laeynx	Notani PN, et al.	1987	Hospital/ population CC	<40~>60	80	2, >2 cups/D,. <2 cups/D	Age and tobacco habits
	Pintos J, et al.	1994	Hospital CC restropective	<54~>75	97	3, Never ≤1, >1 cups/W	Tobacco, alcohol, income, rural residency, 10 dietary variables and consumption of other nonalcohol beverages
	Galeone C, et al.	2010	Pooled data of 9 CC restropective	18–80	1178	3, Never, ≤1, >1/W	Age, sex, race/ethnicity, education, study, cigarette smoking (pack-years), duration of cigar smoking, duration of pipe smoking, alcohol intake, weight, and vegetable and fruit intake.
	La Vecchia C, et al	1992	Population CC restropective	<85	149	2, Nondrinker, drinker	Age, sex, area of residence, education, smoking, and coffee consumption
	Kapil U, et al	2005	Hospital CC restropective	41–80	305	2, Nondrinker, drinker	NA
	Ren JS, et al	2010	Population cohort rrospective	50–71	179	5, None, <1 cup/M, 1–3 cups/M 1–6 cups/W, ≥1 cup/D	Age, sex, tobacco smoking, alcohol drinking, BMI, education, ethnicity, usual physical activity throughout the D, vigorous physical activity and the daily intake of fruit, vegetables, red meat, white meat and calories

C-C – case-control; OP – oropharyngeal; BMI – body mass index; W – week; M – month; D – day.

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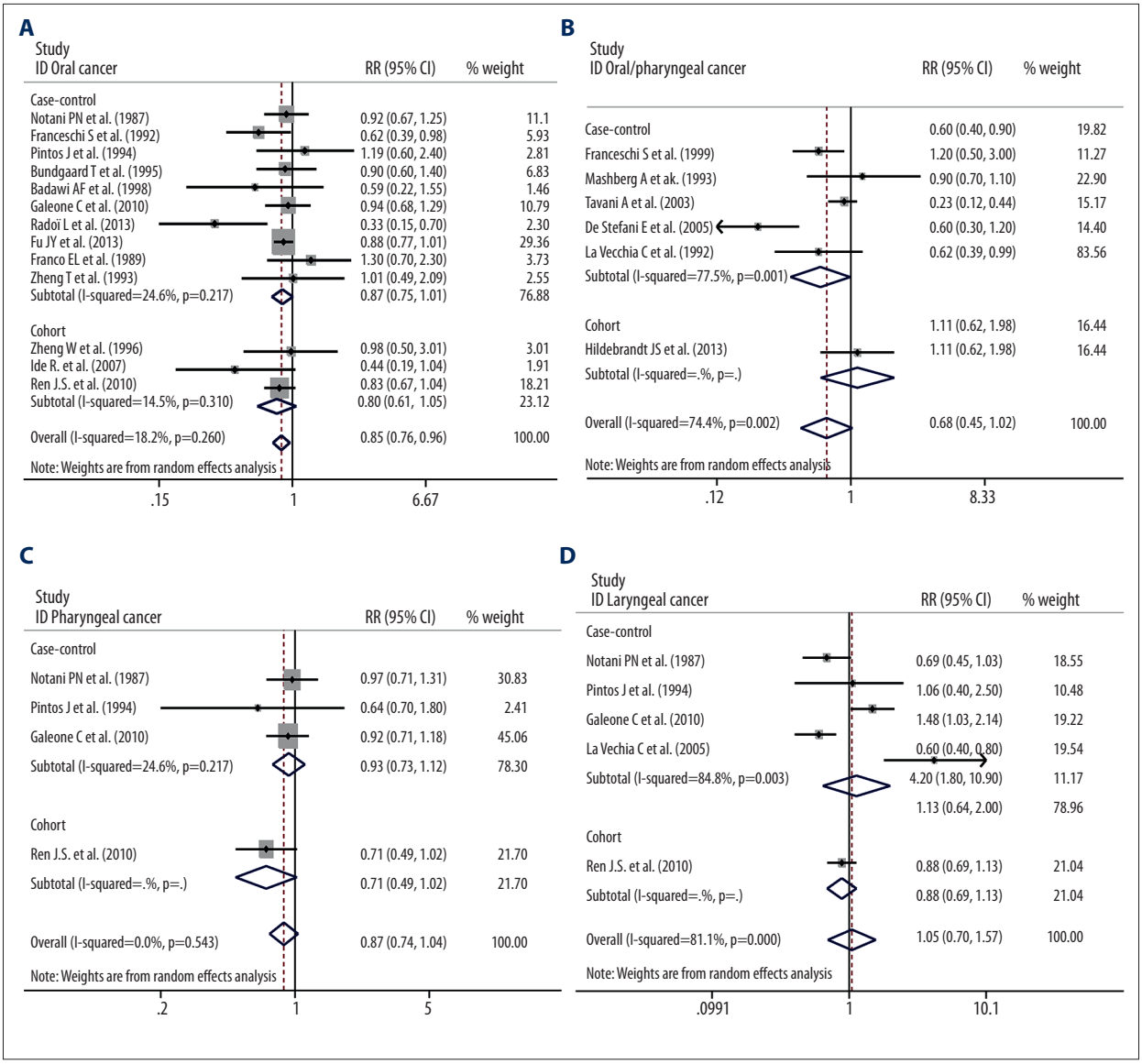

Figure 2. The forest plot for detection of (**A**) tea intake and oral cancer risk, (**B**) tea intake and oral/pharynx carcinoma risk, (**C**) tea intake and pharynx carcinoma risk, and (**D**) tea intake and laryngeal carcinoma risk.

the case-control (RR=0.93; 95% CI 0.76–1.12) nor the cohort study (RR=0.71; 95% CI 0.49–1.02) showed a significant result. Six studies reported the association between tea consumption and oral and pharyngeal carcinoma (Figure 2C). In the general quantitative synthesis, tea intake did not modify the incidence of oral and pharyngeal carcinoma (RR=0.68; 95% CI 0.62–1.98). The results of the subgroup analyses were different. Tea consumption was inversely associated with the oral and pharyngeal carcinoma in the case-control studies (n=5; RR=0.62; 95% CI 0.39–0.99) but not in the cohort study (n=1; RR=1.11; 95% CI 0.62–1.98). The summary RR for laryngeal carcinoma (Figure 2D) was 1.05 (95% CI 0.70–1.57). No significant association was detected in the case-control (n=5; RR=1.13; 95% CI: 0.64-2.00) or the cohort studies (n=1; RR=0.88; 95% CI 0.69–1.13).

Test for heterogeneity

No significant heterogeneity was detected in the included studies for association between tea consumption and risk of oral cancer (I^2=18.2%, P=0.260) or pharyngeal cancer (I^2=0, P=0.543). However, when the effect of tea intake on oral and pharyngeal cancer (I^2=74.4%, P=0.002) and laryngeal cancer (I^2=81.1%, P<0.001) was detected, a significant heterogeneity was detected. The source of the heterogeneity was explored by excluding the included studies one by one; however, no single article influenced the significance of the heterogeneity. The subgroup analysis by study design demonstrated no significant results in exploring the source of heterogeneity.

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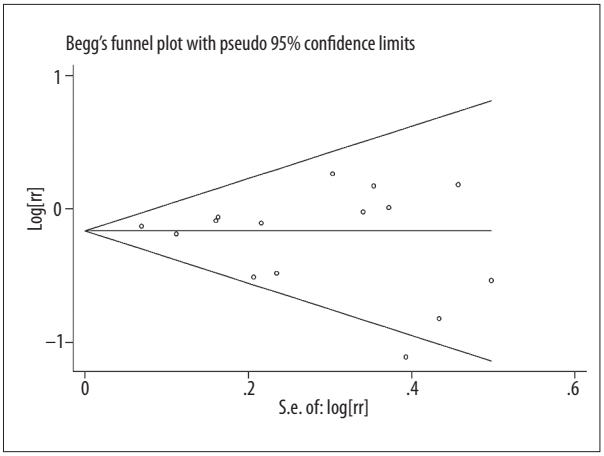

Figure 3. Funnel plot for assessment of publication bias.

Sensitivity analysis and publication bias

The results of the sensitivity analysis suggest that the influence of each individual data set on the pooled RRs was not significant. After excluding the studies with NOS score of less than 6 stars, the results were not affected.

The Begg's funnel plot and the Egger's test showed no evidence of publication bias (oral carcinoma, P=0.398; pharyngeal carcinoma, P=0.407; oral and pharyngeal carcinoma, P=0.473; laryngeal carcinoma, P=0.352) (Figure 3).

Discussion

A total of 11 984 cases in 20 relevant studies were identified in the current meta-analysis and we found a significant reduction in the risk of oral cancer. However, no significant association between tea consumption and pharyngeal, oral/pharyngeal, or laryngeal cancer was detected. When the association between tea drinking and risk of oral cancer was analyzed, the subgroup analyses stratified by study design demonstrated no significant associations in case-control or cohort studies. When only the case-control studies were analyzed, tea consumption was inversely associated with oral/pharyngeal carcinoma. The heterogeneity was significant when the effect of tea intake on risk of oral, pharyngeal, and laryngeal cancer was detected; however, this is understandable considering the heterogeneity in the study designs and data set. The results of the sensitivity analysis and the publication bias detection suggest that the conclusions of this study are quite robust.

In this meta-analysis, tea consumption was inversely associated with the incidence of oral cancer. An inverse association with oral cancer was found in a population-based case-control study with face-to-face interviews and standardized questionnaires [36]. In a case-control study conducted in northern Italy, 102 patients with cancer of the tongue, 104 patients with cancer of the mouth, and 726 control subjects were included, and tea consumption was found to reduce the risk of oral cancer [19]. Among all the 13 included studies for this quantitative synthesis, only 2 studies showed that tea drinking was a protective factor against oral cancer. However, most studies demonstrated a slight but not significant protective effect. In the subgroup analyses stratified by study design, no significant association was detected. We hypothesized that the number of the included articles in each subgroup decreased and thus might increase the confidence interval. In addition, we adopted a random-effects model in this meta-analysis. Considering that no heterogeneity was detected, a random-effects model might partly explain this result.

In general, tea consumption was not associated with the risk of oral/pharyngeal, pharyngeal, or laryngeal carcinoma. In the subgroup analyses in which only the case-control studies were included, tea consumption was inversely associated with oral/pharyngeal carcinoma. However, considering that inherent recall bias existed in the case-control studies, the synthesis of the case-control studies might produce a result with more potential bias, but this result should be interpreted with caution considering the small number of studies in the each subgroup.

A total of 4 studies were included in the meta-analysis of the association between tea drinking and pharyngeal carcinoma and all 4 studies demonstrated a nonsignificant association. Laryngeal cancer is the most common cancer of the head and neck [39]. In this meta-analysis, tea intake was reported to be not associated with laryngeal carcinoma risk. However, a pooled analysis of 9 case-control studies found that tea consumption was associated with a slight but significant increased risk of laryngeal cancer (OR=1.48; 95% CI 1.03–2.14, >1 cup/day *vs.* ≤1 cup/day) [34]. In a hospital-based, matched, case-control study, tea drinking was reported to be laryngeal cancer risk factor (RR=4.2; 95% CI 1.8–10.09, tea drinkers *vs.* nondrinkers) [38]. In a case-control study in northern Italy between 1983 and 1990, a cancer-protective effect of tea was detected as well [20]. Contradictory conclusions might be due to geographical differences, genetic diversity, and bias of the studies. This indicates that more studies are required to offer further evidence of the relationship between tea and cancer risk.

The strengths of the present study are: (1) our analysis included all the studies investigating the association between tea consumption and oral, pharyngeal, and laryngeal carcinoma and involving a total of 11 984 cases in 20 relevant studies, which adds to the strength of the meta-analysis (2) Most of the studies included in this meta-analysis demonstrated a relatively high quality. The results of the sensitivity analysis and the publication bias detection suggest that the conclusions of this meta-analysis are quite robust, which may add strength to the conclusions drawn.

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As with any meta-analysis of observational studies, our study has several limitations. Firstly, most of the studies had a case-control design, and therefore there were recall and selection bias, which are inherent to retrospective studies. Although subgroup analysis by study design was conducted for each cancer, the efficiency was limited by the absence of the cohort studies. Secondly, the included studies failed to provide information on the characteristics of tea, such as cup size, type of tea (green tea or black tea) and duration of tea drinking, which are responsible for the different effects on cancer. Thirdly, several detailed and important analyses are short. For instance, tobacco smoking and alcohol intake are known factors for these cancers. The way in which the tea consumption interacted with these etiological factors should be considered in the subsequent studies. The results of a systematic review strongly suggest that high-temperature beverage drinking increases the risk of esophageal cancer [40]. Tea is usually served as a hot beverage and tea drinking might modify the incidence of the relevant cancers with the effects of the tea content itself.

Conclusions

Tea consumption was associated with a decreased risk of oral cancer, but no association was detected oral/pharyngeal, pharyngeal, or laryngeal cancer. Nevertheless, because of the potential limitations of this meta-analysis, the results should be interpreted with caution and large-sample and well-designed studies are required to confirm our conclusions.

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