Nutrition Research Reviews, page 1 of 10 doi:10.1017/S0954422424000040

Effect of vegetable consumption on risk of gastric cancer: a systematic review and multi-level meta-analysis of prospective studies

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Abstract

Vegetables are known to be beneficial to human health, but the association between vegetable consumption and gastric cancer remains uncertain. To synthesise knowledge about the relationship between vegetable group consumption and gastric cancer risk, update present meta-analyses and estimate associations between vegetable consumption and gastric cancer risk based solely on prospective studies, we perform a PRISMA-compliant three-level meta-analysis. Systematic search identified thirteen prospective studies with fifty-two effect sizes that met all inclusion criteria and no exclusion criteria for our meta-analysis. Pooled risk ratios (RRs) showed a positive association between high vegetable consumption and low gastric cancer risk (pooled RR 0·93, 95% confidence interval 0·90–0·97, $P = 0·06$). In moderator analyses for indicators of gender, region and quantity of vegetable intake, there was no significant difference between subgroups. However, the effect became significant in populations with lower than the minimum risk exposure level (TMREL) of vegetable consumption ($P < 0·05$). Higher vegetable intake is associated with a decreased risk of gastric cancer. This effect may be limited to specific populations, such as ones with lower vegetable consumption. Evidence from our study has important public health implications for dietary recommendations.

Key words: **Vegetable: Gastric cancer: Minimum risk exposure level: Multi-level meta-analysis: Prospective studies**

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Introduction

Modifiable lifestyle risk factors, including diet, are known to be associated with several adverse health outcomes[1]. It is often said that vegetable consumption is beneficial to health. The 2019 Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) estimated that over 400 000 deaths worldwide were attributable to low vegetable consumption[2] and that low vegetable consumption was responsible for a great burden of disease[2]. Gastric cancer is the fifth most common cancer and the fourth leading cause of death from cancer worldwide[3]. According to GLOBOCAN 2020 estimates of cancer incidence and mortality, there were more than 1 million gastric cancer cases in 2020 and more than 700 000 deaths caused by gastric cancer[4]. Thus, evidence-based strategies are needed for reducing the incidence of gastric cancer.

Cancer Prevention 4th edition recommends that people should eat more vegetables to avoid cancer[5]. The association between vegetable consumption and risk of gastric cancer has been explored in many studies, but the strength of this association is not completely agreed upon[6–8]. Meta-analyses pooled study results have estimated the association between vegetable consumption and gastric cancer risk, but many of the meta-analyses were based on case–control studies[9,10], and few were based on prospective studies[11–13]. Most studies were limited to one type of vegetable – most commonly cruciferous and allium vegetables[14–18]. Additional high-level evidence is needed.

An understanding of the direction of association between vegetable consumption and gastric cancer is essential for policy-making and individual health decisions[2]. Therefore, we conducted a systematic review to update existing meta-analyses and estimate associations between vegetable consumption and gastric cancer risk based on prospective studies, with a focus on early modifiable factors that may be able to show good effectiveness at low cost.

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Table 1. Search strategy for PubMed

Batch	Search terms or combinations of the search terms	Results
1	'stomach neoplasms' (MeSH Terms) OR 'stomach cancer' (Title/Abstract) OR 'stomach neoplasm' (Title/Abstract) OR 'stomach tumor' (Title/Abstract) OR ''stomach carcinoma' (Title/Abstract) OR 'gastric cancer' (Title/Abstract) OR 'gastric neoplasm' (Title/Abstract) OR 'gastric tumor' (Title/Abstract) OR 'gastric carcinoma' (Title/Abstract)	135 983
2	'vegetables' (MeSH Terms) OR 'vegetables' (Title/Abstract) OR 'green leafy vegetables' (Title/Abstract) OR 'cruciferous vegetables' (Title/Abstract) OR 'fruits and vegetables' (Title/Abstract)	66 226
3	1 AND 2	605

Methods

The design, implementation, analysis and reporting of our meta-analysis were conducted in accordance with the PRISMA statement (Supplementary Table S1). The systematic review protocol was registered on PROSPERO (CRD42023424878).

Search strategy

Seven literature databases were systematically searched: PubMed, Web of Science, Cochrane Library, SpringerLink, Wiley Online Library, CNKI and Wanfang Med Online. We conducted additional search of the following websites for 'grey literature': System for Information on Grey Literature in Europe, ProQuest research library, China Doctoral Dissertations Full-text Database, and China Master's Theses Full-text Database. Searches were unlimited by time up to 20 March 2023, included English or Chinese literature, and used these MeSH terms and phrases: 'stomach neoplasms' (MeSH Terms) OR 'gastric cancer' (Title/Abstract) OR 'stomach cancer' (Title/Abstract) OR 'stomach neoplasms' (Title/Abstract) OR 'gastric neoplasms' (Title/Abstract) OR 'stomach tumor' (Title/Abstract) OR 'stomach carcinoma' (Title/Abstract) OR 'gastric tumor' (Title/Abstract) OR 'gastric carcinoma' (Title/Abstract)) AND ('vegetables' (MeSH Terms) OR 'vegetables' (Title/Abstract) OR 'green leafy vegetables' (Title/Abstract) OR 'cruciferous vegetables' (Title/Abstract) OR 'fruits and vegetables' (Title/Abstract). Table 1 presents details of the search strings from PubMed.

Two investigators (X.W. and G.Q.) conducted the literature search, and independently reviewed the articles retrieved. Disagreements whether a study met inclusion criteria were resolved by discussion with a third investigator (R.Z.).

Selection criteria and exclusion criteria

We used a PICOS (Population, Intervention, Comparison, Outcomes, Study design) framework to define search strategies and establish eligibility criteria (Table 2). Studies were eligible for inclusion if they met all of the following criteria: (1) the study was prospective; (2) had a total sample size over 100; (3) assessed vegetable consumption; (4) the outcome was defined as the risk of onset of gastric cancer; and (5) the authors reported rate ratio (RR) estimates or hazard ratio (HR) estimates, including 95% confidence intervals (CIs). Studies were excluded if they met any

Table 2. PICOS criteria for inclusion and exclusion of studis

Parameter	Criteria
Population	Adults (human) aged ≥18 years. No restriction on sex, race or ethnicity
Intervention	Vegetables consumption
Control/comparator	Habitual diet poor in vegetables
Outcomes	Onset of gastric cancer
Study design	Prospective studies

of the following criteria: (1) were duplicate publications; (2) were not relevant; (3) were systematic reviews, meta-analyses, meeting abstracts, letters or dissertations without all relevant information; (4) were not prospective studies; and (5) did not report RR or HR and 95% CIs. In case of duplicate publications of the same study, publications with the largest sample sizes or the longest durations of follow-up that provided RR or HR estimates from the same cohort were preferentially included. We used Stanaway and colleague's definition of vegetable consumption[2]. Duplicate records were excluded before screening, and exclusion criteria were applied before inclusion criteria. Figure 1 shows the study flow diagram.

Data extraction and quality assessment

Three researchers (X.W., G.Q. and R.Z.) extracted and verified data from included articles. Extracted data included (a) basic study information: first author, publication year, country of the original study, (mean) age and sample sizes; (b) duration of follow-up and follow-up rates; (c) incidence of gastric cancer; (d) exposure assessment measure and exposure categories; (e) range of exposures and risk estimates and corresponding uncertainty for each exposure category; (f) outcome ascertainment method; and (g) variables for adjusted analyses.

Quality assessment was performed in accordance with the Newcastle–Ottawa Scale (NOS) for observational studies. This scale assigns a maximum of nine points to each study: four for selection of participants, two for comparability and three for outcome assessment. Higher scores indicate better quality. Studies included in the analysis for each risk–outcome pair and study characteristics are presented in Tables 3 and 4, and Supplementary Table S3.

Standardised exposure and estimation of minimum risk exposure level

The theoretical minimal risk exposure level (TMREL) is the level of exposure that, within the range of values that are theoretically possible at the population level, will minimise the risk of all outcomes associated with that risk combined[2]. Based on the standardised method used by Stanaway and colleagues[2], we unified the quantity of vegetable consumption and defined the TMREL.

Statistical analysis

RRs or HRs and their corresponding 95% CIs were considered to be the effect sizes for all studies in our meta-analysis. Effect sizes from multiple adjusted models were included in this study.

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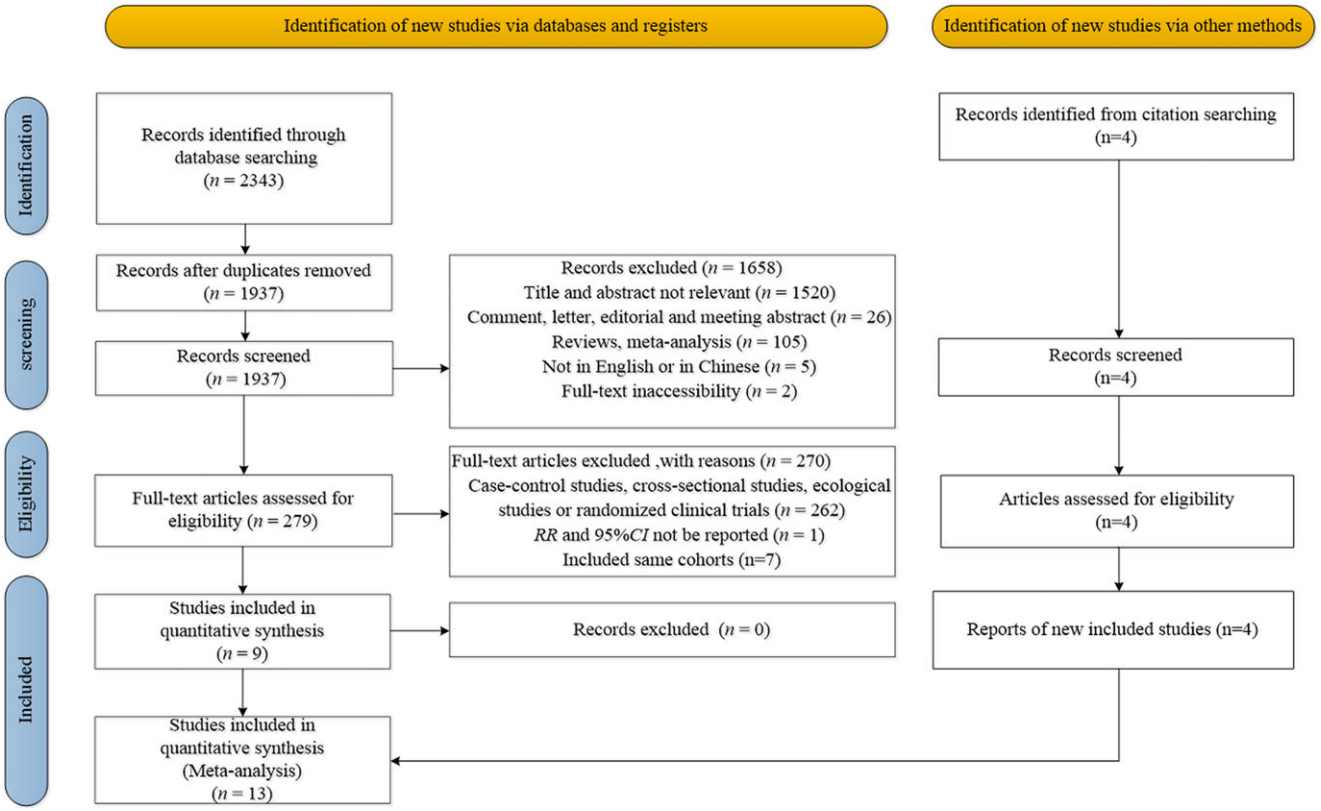

Fig. 1. Flow chart of selection of studies for the meta-analysis.

Table 3. Characteristics of studies included in the meta-analysis

Author and publication year	Methods of exposure measure	Outcome measure	Follow-up rate (%)	Follow-up (years)	Quality score
Nomura *et al.* 1990[19]	Food frequency questionnaire	Surveillance	98·40	19·0	8
Kato *et al.* 1992[20]	A questionnaire survey	Medical records, cancer registry or death certificates	88·70	4·4 (mean)	5
Inoue *et al.* 1996[21]	The self-administered questionnaire	Hospital records, the Aichi Prefectural Cancer Registry and death certificates	Not reported	6·0 (mean)	5
Galanis *et al.* 1998[22]	A short food frequency questionnaire	The Hawaii Tumor Registry	95·00	14·8 (mean)	6
Zhou *et al.* 2005[23]	Food frequency questionnaire	Cancer registries	99·46	13·0	7
Nouraie *et al.* 2005[24]	A questionnaire	The Finnish Cancer Registry	Not reported	12·0	6
Tran *et al.* 2005[25]	A baseline questionnaire	Through monthly visits by village health workers	Not reported	15·0	6
Larsson *et al.* 2006[8]	Food-frequency questionnaire	The national and regional Swedish Cancer registers	Not reported	7·2 (mean)	6
Freedman *et al.* 2008[26]	A questionnaire	State cancer registry databases	Not reported	4·5 (mean)	7
Epplein *et al.* 2010[7]	A comprehensive food frequency questionnaire	Shanghai Cancer Registry and the Shanghai Vital Statistics database and biennial in-home interviews	SWHS: 99·80, 98·70, 96·70; SMHS: 97·60	SWHS: 11·0; SMHS: 6·0	8
Steevens *et al.* 2011[6]	The self-administered questionnaire	The Netherlands Cancer Registry and the nationwide network and registry of histopathology and cytopathology in the Netherlands	≥96·00	16·3	8
Gonzalez *et al.* 2012[27]	Country-specific validated questionnaires	Health insurance records, cancer and pathology hospital registries and active follow-up	Not reported	11·0	7
Shimazu *et al.* 2014[28]	Self-administered food frequency questionnaires	Population-based cancer registries, active patient notification	JPHC I: 82·00, JPHC II: 80·00, MIYAGI: 92·00, JACC: 83·00	Over 10·0	8

Note: SWHS, The Shanghai Women's Health Study, include three stages; SMHS, The Shanghai Men's Health Study; JPHC, Japan Public Health Center-based prospective Study; MIYAGI, The Miyagi Cohort Study; JACC, The Japan Collaborative Cohort Study

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Table 4. Baseline information of studies included in the meta-analysis

Author and publication year	Exposure	Group	Age(years)	Region	Sample size	Incidence rate ((‰)/(per 100,000 person-years))
Nomura *et al.* 1990[19]	Fried vegetables	Both groups: group 1: ≤1 time/week*, group 2: ≥2 times/week	≥45	America	Group 1: 7371, group 2: 617	Age-specific: 45–54, 15·6; 55–64, 74·2; 65–74, 158·8; ≥75, 202·4
Kato *et al.* 1992[20]	Raw vegetables	Three groups: group 1: ≤1–2/month*, group 2: 2–3/week, group 3: daily	All age groups	Asia	5395	Group 1: 314·4, group 2: 260·9, group 3: 210·4
Inoue *et al.* 1996[21]	Raw vegetables	Three groups: group 1: rarely*, group 2: occasionally, group 3: daily	All age groups	Asia	5373	Group 1: 370·6, group 2: 221·0, group 3: 223·2
Galanis *et al.* 1998[22]	Raw vegetables	Both groups: group 1: 0–6 times/week*, group 2: 7 or more times/week	46·4 ± 16·6	Asia	Group 1: 3564, group 2: 8343	Group 1†: 9·3, group 2†: 9·0
Zhou *et al.* 2005[23]	Fresh vegetables	Both groups: Group 1: daily*, group 2: rarely	Male: 46·12 ± 11·90; female: 46·94 ± 12·72	Asia	Group 1: 52 848, group 2: 2259	Group 1: 51·5, group 2: 87·8
Nouraie *et al.* 2005[24]	Total vegetables	Quartiles (g/d): group 1: ≤66*, group 2: 67–100, group 3: 101–147, group 4: ≥148	57 (50, 66)	Europe	27 110	8·12†
Tran *et al.* 2005[25]	Fresh vegetables	Quartiles (times/year): group 1: ≤549*, group 2: 549–732, group 3: 732–915, group 4: ≥915	52 (median)	Asia	29 584	49·1†
Larsson *et al.* 2006[8]	Total vegetables	Quartiles (servings/d): group 1: ≤1·0*, group 2: 1·1–1·4, group 3: 1·5–2·4, group 4: ≥2·5	45–83	Europe	82 002	23·5
Freedman *et al.* 2008[26]	Total vegetables	Median intake of those in quintile (daily servings per 1000 KJ) Group 1: 0·71*, group 2: 1·15, group 3: 1·56, group 4: 2·08, group 5: 3·15	62 (median)	America	98 160	18·0
Epplein *et al.* 2010[7]	All vegetables	Quartiles (g/d): group 1: ≤179·5*, group 2: 179·5–261·3, group 3: 261·3–373·7, group 4: ≥373·7	Male: cohort 55·2 (9·7), case 62·1 (9·8); female: cohort 52·5 (9·1), case 58·2 (9·0)	Asia	Male: 59 247, female: 73 064	Male†: 2·23, female†: 2·82
Steevens *et al.* 2011[6]	Total vegetables	Median intake of those in quintile (g/d): group 1: 104*, group 2: 146, group 3: 181, group 4: 222, group 5: 297	Subcohort 55·2 (9·7), GCA 61·4 (4·0), GNCA 62·5 (4·1)	Europe	120 852	GCA: 306·9, GNCA: 918·7
Gonzalez *et al.* 2012[27]	Total vegetables	Male: quintile (g/d) Group 1: 0–83*, group 2: 83–126, group 3: 126–182, group 4: 182–282, group 5: 282–2310 Female: quintile (g/d) Group 1: 0–105*, group 2: 105–156, group 3: 156–219, group 4: 219–315, group 5: 315–2979	51·2, mean	Europe	477 312	13·0

Nutrition Research Reviews

Table 4. (*Continued*)

Author and publication year	Exposure	Group	Age(years)	Region	Sample size	Incidence rate (‰)/(per 100,000 person-years))
Shimazu *et al.* 2014[28]	Total vegetables	Male: quintile of intake (g/d) JPHC I: group 1: <100*, group 2: 100–136, group 3: 136–179, group 4: 179–227, group 5: >227; JPHC II: group 1: <21*, group 2: 21–37, group 3: 37–53, group 4: 53–76, group 5: >76; MIYAGI: group 1: <39*, group 2: 39–64, group 3: 64–90, group 4: 90–129, group 5: >129; JACC: group 1: <65*, group 2: 65–92, group 3: 92–120, group 4: 120–157, group 5: >157 Female: quintile of intake (g/d) JPHC I group 1: <115*, group 2: 115–153, group 3: 153–193, group 4: 193–233, group 5: >233; JPHC II group 1: <27*, group 2: 27–40, group 3: 40–55, group 4: 55–77, group 5: >77; MIYAGI: group 1<54*, group 2: 54–81, group 3: 81–109, group 4: 109–152, group 5: >152; JACC: group 1: <80*, group 2: 80–108, group 3: 108–135, group 4: 135–170, group 5: >170;	JPHC I 40–59, JPHC II 40–69, MIYAGI 40–64, JACC 40–79	Asia	Male 87 771 Female 103 461	143·0

Note: GCC, gastric cardia cancer; GNCC, gastric noncardia cancer; GCA, gastric cardia adenocarcinoma; GNCA, gastric noncardia adenocarcinoma; JPHC, Japan Public Health Center-based prospective Study; MIYAGI, The Miyagi Cohort Study; JACC, The Japan Collaborative Cohort Study.
* Reference group.
† Cumulative incidence.

Values from each study and corresponding standard errors were transformed into natural logarithms to stabilise variances and normalise distributions. Pooled RRs and corresponding 95% CIs were estimated using a random effect model, weighted by the inverse of the variance. Heterogeneity among studies was estimated using the I^2 statistic, with a P value <0·10 considered significant. A forest plot was used to visualise pooled effect size. We used a three-level meta-analysis model with random effects due to the hierarchical data structure. A three-level model accounts for sampling variance (level 1), variance within-study (level 2) and variance between studies (level 3). If there was heterogeneity at level 2 or level 3, we used moderator analyses to examine the heterogeneity. In addition to the three-level modelling, we also developed a reduced model (two-level model) by removing the second-level unit (within-study level) or the third-level unit (between-study level) for model diagnostics and selection. Potential publication bias was visualised with a funnel plot. Egger's linear regression test was used to measure asymmetry of the funnel plot, with a P value <0·10 considered significant. Effects of outliers was examined with sensitivity analysis. Outliers were removed and the overall effect of the association between vegetable consumption and onset risk of gastric cancer was re-estimated. We re-estimated the overall effect of all aggregated effect sizes by applying the aggregate function.

All analyses were performed by the open-source R program (Version 3.6.2; R core team, R Foundation for Statistical Computing, Vienna, Austria). The 'meta' package, 'metafor' package and 'dmetar' package were used to perform analyses and visualisation of results.

Results

Study selection and characteristics

Search retrieved 2343 articles from the eleven databases; 2330 were excluded as shown in Fig. 1. Thirteen studies met all inclusion criteria and no exclusion criteria; all were published between 1990 and 2014, were prospective and were included in our meta-analysis. Tables 3 and 4 present relevant characteristics of the included studies. Four of the studies were conducted in Japan, three in China, two in the United States, one in several European countries, one in Finland, one in Sweden and one in the Netherlands. All studies used questionnaires to assess vegetable consumption. Six reported total vegetable consumption, three reported raw vegetable consumption, two reported fresh vegetable consumption, one reported fried vegetable consumption and one reported all vegetable consumption. The total sample size of the included studies was 1 244 333, ranging from 5373 to 477 312 subjects per study. Follow-up periods ranged from 4·4 years to 19 years. Seven studies reported moderate or good compliance; six did not report follow-up rates. NOS was used to assess the quality of included articles. Details of the NOS evaluation are presented in Supplementary Table S2.

Overall mean effect size

A significant overall association was found between vegetable consumption and risk of gastric cancer (pooled RR 0·93, 95% CI

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Table 5. Results of the moderator analyses

Moderator	Cohen's *d* (95% CI)	$F(df_1, df_2)$	*P*
Region		$F(2,49) = 2{\cdot}2661$	0·1145
America (re)	0·07 (−0·21, 0·35)		
Asia	−0·15 (−0·45, 0·15)		
Europe	−0·29 (−0·60, 0·02)		
Gender		$F(2,49) = 0{\cdot}8089$	0·4512
Female (re)	−0·25 (−0·51, 0·01)		
Male	0·23 (−0·14, 0·60)		
Both	0·13 (−0·15, 0·41)		
Quantity		$F(1,50) = 0{\cdot}1058$	0·7463
Lower than TMREL (re)	−0·14 (−0·24, −0·03)*		
TMREL or higher than TMREL	0·02 (−0·10, 0·13)		

Note: re, reference; TMREL, the minimum risk exposure level.
* $P < 0.05$.

0·90–0·97, $P = 0{\cdot}06$, results based on $k = 52$ effect sizes). Figure 2 shows the pooled analyses forest plot. Heterogeneity was low and acceptable ($I^2 = 25\%$).

Multi-level meta-analyses

The thirteen studies reported fifty-two effect sizes of the association between vegetable consumption and risk of gastric cancer. The full three-level model that captured three-level data heterogeneity had the best goodness-of-fit with the lowest Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) (AIC in full model −10·9102 and BIC in full model −5·1147). In the three-level model, we found that 63·00% of the total variance could be attributed to within-study differences and 34·35% of the total variance could be attributed to between-study differences (Supplementary Figure S1).

Moderator analyses

Table 5 presents the results of moderator analyses that were performed to determine potential variables that can explain the variance at level 2 or level 3. We estimated a theoretical minimal risk exposure level of 182–204 g/d. Moderator analyses for gender, region and quantity of vegetables consumed showed that their indicators were not significant ($P > 0{\cdot}05$), implying no significant differences between subgroups. However, moderator analysis for lower than the TMREL of vegetable consumption showed that subgroup-specific effects were significant ($P < 0{\cdot}05$). The mean effect of more vegetable consumption was lower than the mean effect of lower vegetable consumption among a population consuming vegetables at levels less than the TMREL.

Publication bias

Supplementary Figure S2 shows funnel plots of the effect sizes. Visual inspection of the relationship between effect sizes and their corresponding SEs shows asymmetry. SEs is standard deviation of statistic, which describes the sampling error. The Egger's test was significant ($P = 0{\cdot}04$), indicating presence of publication bias.

Sensitivity analyses

We identified two influential outliers. The effect size estimate without influential outliers remained significant for the association between vegetable consumption and risk of gastric cancer (pooled RR 0·92, 95% CI 0·88–0·95, results based on $k = 50$ effect sizes). Single effect sizes from each study were aggregated using the aggregate function and the association between vegetable consumption and the risk of gastric cancer remained significant (pooled RR 0·92, 95% CI 0·85–0·99) (Supplementary Figure S3).

Discussion

In our multi-level meta-analysis study, we found that (1) there is an association between vegetable consumption and risk of gastric cancer (pooled RR 0·93, 95% CI 0·90–0·97, $P = 0{\cdot}06$, results based on $k = 52$ effect sizes) in that greater vegetable consumption is associated with lower gastric cancer risk and (2) moderator analysis for the quantity vegetable consumption showed that subgroup-specific effects are significant ($P < 0{\cdot}05$) in populations with vegetable consumption lower than the vegetable consumption TMREL.

Similar studies

Our study results are similar to some but not all comparable studies. Some meta-analyses found no association between vegetable consumption and gastric cancer risk[9,12,13]. Several factors may explain the discrepancy between our study and these studies. Study design options may have played a key role in this association[12]. Duration of follow-up time may play a role, as one meta-analysis found that the association existed only when considering studies with longer follow-up[12]. Adjusting variables may play a role, as the association became significant only after adjusting total energy intake in one study[11]. The specific histological subtype of gastric cancer may play a role, as the association may only exist in specific histological subtypes of gastric cancer[29]. Vegetable preparation methods may have led to inconsistency of studies[9]. For example, pickled vegetables are salty and high salt intake may increase risk of gastric cancer[9,30]. Such an increase has been confirmed in two meta-analyses, with one finding a potential dose–response relationship[9,30].

Potential mechanisms

The association between vegetable consumption and gastric cancer that our and other studies observed may be explained by several mechanisms. Vegetables contain antioxidant components including vitamin C, vitamin E, carotenoids, phenolics and folate[8,23,28,31]. Bioactive chemicals found in vegetables can inhibit formation of N-nitroso compounds[22,23,27]. Vegetables can play an anti-cancer role by scavenging potentially mutagenic free radicals[8,28]. Vegetables may counteract DNA damage caused by *Helicobacter pylori*, which is a well-known risk factor for gastric cancer[28].

Moderating effect of vegetable consumption quantity

Diets low in vegetables are a leading risk factors for cancer[32]. *The Lancet* EAT Commission recommends that people consume

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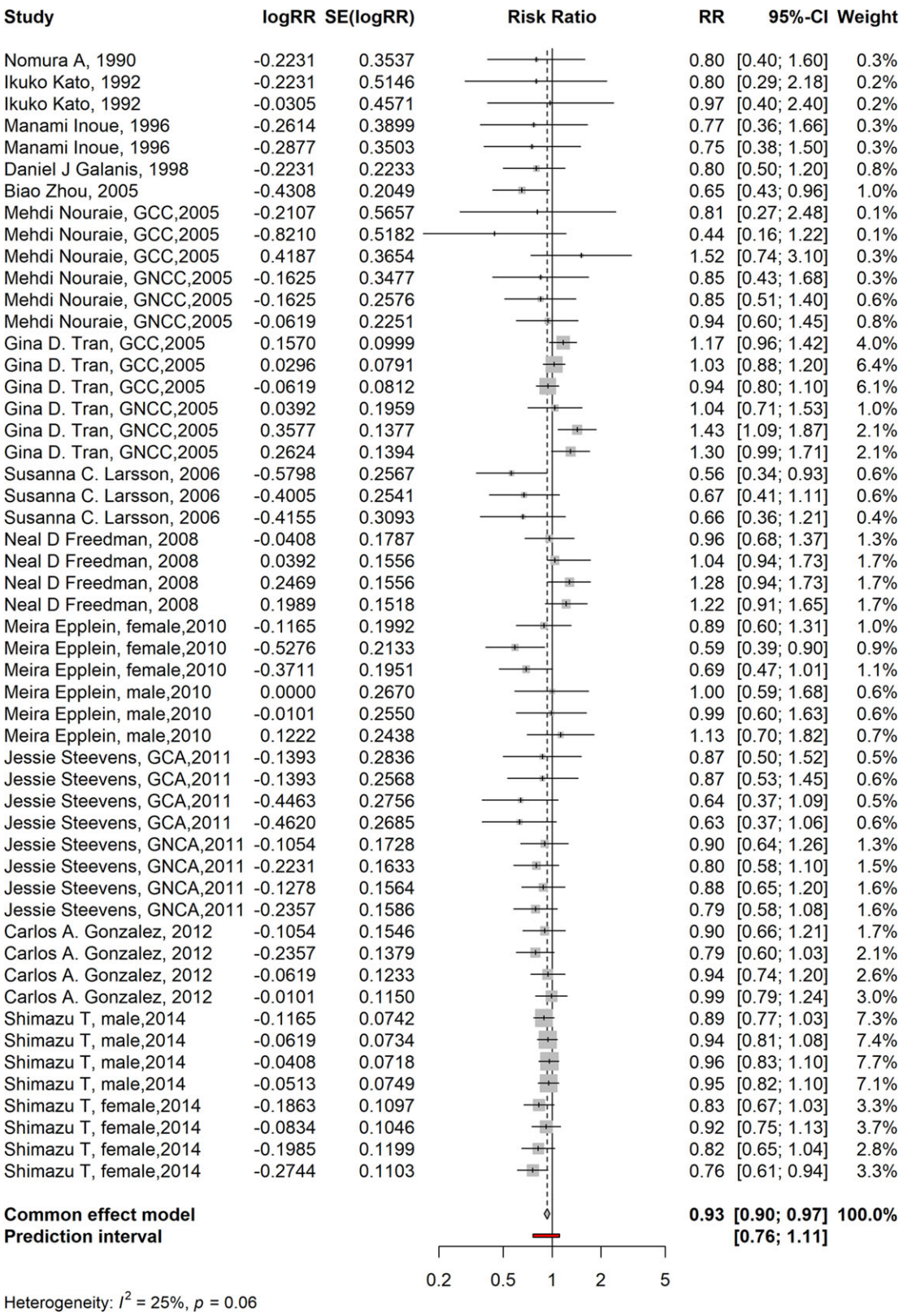

Fig. 2. Forest plot of associations between vegetables consumption and gastric cancer risk. GCC, gastric cardia cancer; GNCC, gastric noncardia cancer; GCA, gastric cardia adenocarcinoma; GNCA, gastric noncardia adenocarcinoma.

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300 g of vegetables per day to promote individual human health[33]. One study estimated the TMREL of vegetables on oral cancer, nasopharyngeal cancer, cancer of the pharynx and oropharynx, laryngeal cancer, ischaemic heart disease, haemorrhagic and non-ischaemic stroke[32], finding that the mean TMREL was 400 g/d of vegetable consumption[32]. Meta-analysis of prospective studies on the intake of 550–600 g/d of vegetables found a 12% reduction in the relative risk of cancer[34]. However, data regarding vegetable consumption and gastric cancer risk are less clear-cut. Additional quantitative information of vegetable consumption and gastric cancer should be provided in future studies. Our TMREL estimate of 182–204 g/d is in line with recommendations. We found that a protective effect was significant in populations with levels of vegetable consumption below the TMREL, raising the intriguing possibility that the protective effect of vegetables on gastric cancer has a threshold[8,28]. We found that consuming vegetables above a certain level was associated with no additional benefit. Supporting this notion and consistent with our results, a recent meta-analysis found that 130–400 g vegetable consumption per day has a protective effect[11]. Based on the totality of evidence, we suggest that consumption of at least 200 g of vegetables per day is beneficial, and that more vegetable consumption is better.

Study evidence compared with protective effect of fruit consumption

Current evidence suggests that vegetable consumption is inversely associated with gastric cancer risk. Fruits also have potential anti-cancer effects and have in common with vegetables potential mechanisms for a protective effect including, antioxidation, radical-scavenging activity and counteraction of DNA damage[11]. Well-regarded organisations recommend fruit and vegetable consumption, ranging from at least 400 g/d by the World Cancer Research Fund (WCRF) and the World Health Organization, to 500 g/d in Sweden and 640–800 g/d in the United States[34]. Mounting evidence confirms that fruit consumption has a protective effect on gastric cancer[11,35]. The TMREL for fruit was 200 g/d and 300 g/d in two studies, respectively. TMREL of vegetables varies by study, as does that of fruits[11,35]. Socio-economic status, natural environment and dietary pattern all have effects[35]. The TMREL of population with affordable and available access to fruits and vegetables is lower than in populations with less access. These factors should be considered when referencing TMRELs.

Strengths and limitations

Our study has several strengthens. First, total effect size was determined from only prospective studies. Recall bias and selection bias would therefore be minimised in our meta-analysis. Second, the association between vegetable consumption and gastric cancer was quantified by introducing a TMREL.

There are several limitations to our study. We only searched for studies published in English or Chinese. Studies published in other languages were not included in this meta-analysis. Second, information on *H. pylori* infection was lacking. Third, a food frequency questionnaire was used to estimate vegetable intake in most studies, which could not capture the actual intake amounts. Only baseline dietary information was used in the study. Fourth, although some potential confounders were adjusted for in each study, it is likely that some residual confounding remains due to unmeasured factors. Fifth, the cut-off values of vegetable consumption varied among studies, challenging uniformity in the meta-analysis. Sixth, only dietary information was collected, in the study and dietary supplement information was not collected. Finally, information on gastric cancer histological subtype was not collected in this study. Therefore, the subgroup analysis for the histological subtype was not able to be performed.

Implications

Evidence from our study supports recommendations increase vegetable consumption. Increasing vegetable consumption will lead to individual-level health benefits and may have environmental benefits. The present evidence helps justify more robust effort and policies to promote increasing vegetable consumption to reduce gastric cancer risk. Public policies should be issued, for example, to publicise the benefits of vegetable consumption to the public, encourage restaurants to sell more vegetable products, and make more kinds of vegetables available to the public. Societies and individuals can reduce the risk of gastric cancer by increasing vegetable consumption.

The pooled RR was 0·93 (95% CI 0·90–0·97), which may be considered not very strong but meaningful at a population level. More prospective studies with large sample size will be needed to further confirm this association.

Supplementary material

The supplementary material for this article can be found at https://doi.org/10.1017/S0954422424000040.

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Data availability

All reported data are available in the manuscript. The analytic code is available upon request.

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Competing interests

No conflict of interest to declare.

Authorship

W.X., Q.G., Z.R., F.Z. and W.J. contributed to the conception and design of the systematic review and meta-analysis. S.J. and K.C. were involved in the acquisition and analysis of the data. L.Y., C.L. and S.J. interpreted the results. W.X. and Q.G. drafted this paper. All authors provided critical revisions of the meta-analysis and approved submission of the final manuscript.

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