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Dietary inflammatory index and the risk of esophageal cancer: a systematic review and meta-analysis

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Abstract

Background and aim It is well-recognized that inflammation is an adaptive pathophysiological response in many types of cancer. Research on nutrition's critical role in inflammation, a risk factor for all forms of cancer, is growing. The dietary inflammatory index (DII) was created lately to assess if a diet is pro- or anti-inflammatory in terms of inflammation. Indeed, several studies have demonstrated the correlation between DII and the risk of several cancer types. This meta-analysis set out to look into the relationship between DII and the different forms of esophageal cancer.

Method PubMed, Cochrane library, Embase, Scopus, and Web of Science databases were searched up to May 2024 to retrieve relevant articles. RAYYAN intelligent tool for systematic reviews was incorporated for the screening of studies. Original articles written in English Studies that investigated the inflammatory index of diet in individuals who developed esophageal cancer were included in this study.STATA v18 software was used to conduct the meta-analysis. Egger's test for publication bias assessment was implemented. Newcastle Ottawa scale was used to evaluate the qualities of the included studies. A plot digitizer was used to extract digital data.

Result A total of 13 studies were included in the systematic review, with 6 studies contributing to the meta-analysis, comprising 10,150 participants. The participants were categorized into high and low DII groups, with the low DII group ($n = 3,403$) serving as the reference. The meta-analysis demonstrated a significant association between high DII and increased risk of esophageal cancer. Specifically, individuals in the high DII group were 29% more likely to develop esophageal cancer, with a pooled odds ratio (OR) of 1.29 (95% Confidence Interval [CI]: 1.16–1.43), as calculated using a random-effects model. Moderate heterogeneity was observed ($I^2 > 50\%$). Egger's test indicated evidence of publication bias ($p < 0.05$). Subgroup and sensitivity analyses confirmed the robustness of this association across populations and study designs.

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Conclusion our study concludes that a higher level of DII is associated with a higher risk of esophageal cancer development. This study suggests that modifying inflammatory properties of dietary patterns can reduce the risk of incidence of esophageal cancer.

Keywords DII, Dietary inflammatory index, Esophageal cancer

Introduction

The Dietary Inflammatory Index (DII) is a tool developed to assess the inflammatory impact of an individual's diet. It evaluates dietary components based on their anti- or pro-inflammatory properties, assigning them a score from anti- to pro-inflammatory [1]. The DII plays a pivotal role in elucidating the association between dietary patterns and the risk of developing various types of cancer. Several studies have indicated that a higher DII score is correlated with a greater risk of developing a range of cancers, including colorectal cancer, esophageal cancer, breast cancer, and prostate cancer [2].

The estimated prevalence of esophageal cancer in the United States is approximately 22,370 new cases diagnosed annually, with an estimated 16,130 deaths from this disease in 2024. Esophageal cancer is the eleventh leading cause of cancer death in the United States, with a death rate of 3.7 per 100,000 men and women per year based on 2018–2022 deaths [3].

Individuals afflicted with this condition may experience difficulty or pain when swallowing solid foods, which may extend to liquids as the cancerous mass expands within the esophagus. Other symptoms may include progressive weight loss, nausea, vomiting, loss of appetite, chest pain, and hoarseness [4].

Chronic, low-grade inflammation resulting from certain dietary patterns can create a favorable microenvironment for tumorigenesis by triggering a cascade of cellular and molecular changes. Pro-inflammatory diets often elevate the production of cytokines (e.g., TNF-α, IL-6) and reactive oxygen species, which can damage DNA, promote mutations, and disrupt key regulatory pathways involved in cell proliferation and apoptosis. Simultaneously, sustained inflammation can epigenetically alter tumor-suppressor genes, further accelerating the malignant transformation. This pro-inflammatory state also supports angiogenesis, enabling tumors to secure nutrients and oxygen for continued growth, and fosters immune evasion, as persistent inflammatory signals can inhibit effective anti-tumor immune responses. Collectively, these processes underscore the critical role of diet-induced inflammation as a driver of cancer risk, highlighting the importance of adopting anti-inflammatory dietary strategies to help mitigate carcinogenesis [5, 6].

The relationship between esophageal malignancy and the Dietary Inflammatory Index (DII) has been the subject of several studies, demonstrating a significant correlation between dietary inflammation and the risk of esophageal cancer. The DII, a novel index that quantifies the inflammatory potential of diet, has been widely utilized to assess the risk of various diseases, including esophageal cancer [7]. The significance of investigating the association between DII and esophageal cancer lies in its potential as a predictive tool for risk. By evaluating the DII, healthcare professionals and researchers can assess an individual's dietary inflammation and guide interventions to reduce the risk of esophageal cancer [7]. Herein, we aimed to conduct a systematic review and meta-analysis on the relationship between the DII and the risk of esophageal cancer.

Methods

The current study is a systematic review and meta-analysis that adheres to the principles outlined in the PRISMA checklist [8]. The study protocol has been registered within the Open Science Framework (OSF) (https://doi.org/10.17605/OSF.IO/C6AP3).

Search strategy

A search of related studies published through February 2024 was conducted primarily in the PubMed, Web of Science (WoS), Scopus, Cochrane library and Embase databases. The leading search terms were as follows: ("dietary inflammatory index" OR "DII" OR "inflammatory index" OR "inflammatory foods" OR "inflammatory diet" OR "pro-inflammatory diet" OR "diet-related inflammation") AND ("esophageal cancer" OR "esophageal neoplasms" OR "esophageal malignancy" OR "esophageal carcinoma"). Additionally, the reference lists of pertinent articles were manually reviewed (Tables 1 and 2).

Inclusion and exclusion criteria

We included studies in our systematic review and meta-analysis according to the following criteria: Original articles written in English Studies that investigated the inflammatory index of diet in individuals who developed esophageal cancer.

The following criteria were used to exclude studies from our study: Not original articles such as systematic reviews and meta-analyses**,** case reports and case series,

Table 1 Search strategies and result of the searching procedure

Data base	Search strategy
PubMed Embase Cochrane library	((("dietary inflammatory index"[Title/Abstract]) OR (DII[Title/Abstract])) OR ("inflammatory index"[Title/Abstract])) AND ((("esophageal cancer"[Title/Abstract]) OR ("esophageal neoplasms"[Title/Abstract])) OR ("esophageal malignancy"[Title/Abstract]))
WOS	1: ((TS = ("dietary in flammatory index")) OR TS = ("DII")) Results: 4618 2: ((TS = ("esophageal cancer")) OR TS = ("esophageal neoplasms")) OR TS = ("esophageal malignancy") Results: 33,257 3: #1 AND #2
Scopus	(TITLE-ABS- KEY ("dietary inflammatory index") OR TITLE-ABS-KEY ("DII") OR TITLE-ABS-KEY ("inflammatory index")) AND (TITLE-ABS-KEY ("esophageal cancer") OR TITLE-ABS-KEY ("esophageal neoplasms") OR TITLE-ABS- KEY ("esophageal malignancy"))

conference letters, expert opinions, non-English articles, and Studies conducted on animals and non-English studies.

Based on PICOs frame woFrk the eligibility criteria was as follow:

Population (P)

Adults (no specific age or sex restrictions) from any geographic region.

Studies including individuals with or without esophageal cancer, where dietary data relevant to the Dietary Inflammatory Index (DII) were collected.

Intervention (I)

Exposure to dietary patterns or components measured using the Dietary Inflammatory Index (DII).

Could include assessment of pro-inflammatory vs. anti-inflammatory diets and their relationship to cancer risk.

Comparison (C)

Groups with differing DII levels (e.g., high DII vs. low DII).

Potential comparisons between DII and other inflammatory indexes (e.g., Systemic Inflammatory Index, SII).

Outcome (O)

Incidence, prevalence, or risk of esophageal cancer.

Measures of association (e.g., odds ratio, risk ratio) relating DII levels to esophageal cancer risk or progression.

Study design (S)

Original, peer-reviewed research (e.g., cross-sectional, case-control, cohort studies) published in English.

Excluded: non-original research (reviews, meta-analyses), animal studies, conference abstracts, case reports, editorials, or expert opinions.

Data extraction and quality assessment

Three individuals were responsible for extracting and screening data from the included studies. The extraction and screening were conducted per the established criteria and guidelines, thus minimizing the likelihood of errors and ensuring the quality of the data collected. Independent reviewers screened the selected articles based on their titles and abstracts using the RAYYAN intelligent tool for systematic reviews.

The data from the included articles were extracted in the following manner: author's name, publication year, country in which the study was conducted, total number of participants, number of cases, number of controls, total number of cases and controls for both female and male participants, Body Mass Index (BMI), DII amount of alcohol consumption, age and education levels of the participants, odds ratio and confidence interval.

The Newcastle–Ottawa Scale (NOS) for cross-sectional and case–control studies was utilized to evaluate the risk of bias in individual studies.

Statistical analysis

A meta-analysis was conducted using data on DII as mean ± SD and the Odds ratio of esophageal cancer development. A random effects model calculates The OR and 95% confidence intervals (CIs). A random effects model was also used to combine the study-specific Standardized Mean Difference (SMD) to determine the pooled estimate of the difference in DII between the case and control groups. Heterogeneity was assessed using the Chi-square and I-square tests. A subgroup analysis was performed to investigate the factors contributing to heterogeneity. Data points from graphical representations in studies were extracted using WebPlot Digitizer (Automeris LLC, Frisco, Texas). All statistical analyses were two-tailed, with significance at a P value < 0.05.

Publication bias assessment

The study examined publication bias using Egger's regression, and when Egger's regression identified significant

Table 2 Summary characteristics of included studies

Author	Year	Country	Population	Gender (M/F)	Meanage	DII/SII/SIS	Follow-upperiod	Overallsurvival	BMI	Education years	Alcoholuse	Findings
ABE M, et al. [9]	2018	Japan	1729 (case: 433 control: 1296)	M: 1498 F: 231	60 years	DII stage & case/control: 1(−4.31 – −1.03) 78/328, 2(− 1.03 – −0.11) 114/323, 3 (0.11–0.57) 110/321, 4(0.57–2.02) 131/324	NA	NA	NA	NA	1282	Higher DII is associated with developing EC.
ACCARDI G, et al. [10]	2019	Italy	1047 (case: 304 control: 743)	M: 90.5% of cases & 79.8% of controls	60 years	DII Cases: −0.501 controls: -0.999 p=0.003	24 years	NA	NA	NA	NA (but present in subgroups)	Higher DII scores in elderly than in middle-aged individuals.
SHIVAPPA N, et al. [11]	2017	USA	480 (case: 224 control: 256)	M: 189 of cases	64.3 years	DII Mean: 1.7 SD: 1.7 p<0.001	NA	NA	Mean: 28.6 SD: 4.9 p=0.001	Mean: 10.7 SD: 2.6 p<0.001	Mean: 20.6 SD: 14.1 p=0.01	High E-DII scores were associated with borderline increase in odds of esophageal adenocarcinoma (OR 2·29; 95 % CI 1·32, 3·96).
TANG L, et al. [12]	2018	China	639 (case: 359 control: 380)	M: 529 F: 110	61 years	DII Cases: −0.35 controls: −1.41	1 year	NA	24.1	*Case;control* None/primary: 183;136 secondary: 140;191 tertiary: 36;53	*Case;control* 166;193	higher DII scores were associated with esophageal cancer risk (ORQuartile 4 v. 1 2·55; 95 % CI 1·61, 4·06; Ptrend < 0·001)

Table 2 (continued)

Author	Year	Country	Population	Gender (M/F)	Meanage	DII/SII/SIS	Follow-upperiod	Overallsurvival	BMI	Education years	Alcoholuse	Findings
CHANG L, et al. [13]	2022	China	67	M: 67 F: 2	60 years	NA	NA	The 1-year OS rates of patients in the low and high CONUT groups were 76.19% and 51.85%.	21.87	NA	46	The CONUT score and SII, neutrophil-to-lymphocyte ratio were an independent prognostic factor for overall survival ($P<0.05$). Furthermore, among patients treated with ICI, a high CONUT score was associated with a significantly worse pro-gression-free survival (PFS) and overall survival compared with a low CONUT group.
JANG J, et al. [14]	2023	China	160	M: 122 F: 38	Median of 70 years	Median SII: 1036.6		Median survival time was 12 months (range from 8 to 35 months) in the high group and 25 months (range from 21 to 36 months) in the low group	NA	NA	NA	Combination analysis observed that SIIlow + TILhigh had the best prognosis of all combinations, with a median OS and PFS of 36 and 22 months, respec-tively. The worst prognosis was identified as SII-high + TILlow, with a median OS and PFS of only 8 and 4 months.

Table 2 (continued)

Author	Year	Country	Population	Gender (M/F)	Meanage	DII/SII/SIS	Follow-upperiod	Overallsurvival	BMI	Education years	Alcoholuse	Findings
JIANG Z, et al. [15]	2021	China	121	M: 91 F: 30 of both groups	EOF 63.37 ± 7.49 LOF 63.74 ± 7.22	(CRP, 47.58 ± 25.72 VS 60.87 ± 30.26, p = 0.01; IL-6, 19.34 ± 12.67 VS 26.19 ± 10.73, p = 0.002) and POD 30 (CRP, 6.13 ± 13.25 VS 13.57 ± 18.96, p=0.013; IL-6, 5.86±6.34 VS 10.35±5.82, p=0.0001)	NA	NA	EOF 22.69 ±2.1 LOF 23.12 ± 2.61	NA	EOF 41, LOF 31	The postoperative nutritional index (ALB, PA, TRF, Hb) and immune index (IgA, IgG, IgM) of the EOF group were higher than those of the LOF group ($p < 0.05$), and the inflammatory indicators (CRP, IL-6) of the EOF group were significantly lower than those of the LOF group ($p < 0.05$). Moreover, postoperative T12- SMA variation and QLQ-C30 scores of the EOF group were higher than those in LOF group ($p < 0.05$).
HUANG Y, et al. [16]	2015	China	327	M: 276 F: 41	59.3 ± 7.8 years	CRP (mean ± SD, mg/L) 9.1±14.9	NA	NA	NA	NA	NA	The area under the curve (AUC) was 0.645 for CRP.

Table 2 (continued)

Author	Year	Country	Population	Gender (M/F)	Meanage	DII/SII/SIS	Follow-upperiod	Overallsurvival	BMI	Education years	Alcoholuse	Findings
HUANG C, et al. [17]	2023	China	166	M: 117 F: 49	Median of 70 years	SIS=0: 79 SIS=1:71 SIS=2: 16 SIS(HR 95% CI 1.58(1.21–2.08))	NA	OS of SIS = 0, SIS = 1 and SIS = 2 was 28.0 ± 2.9, 16.0 ± 2.8 and 10.0 ± 7.0 months.	Median of 18.5	NA	NA	The AUCs of NLR, PLR and SII were significantly lower than the AUC of SIS, meaning that SIS showed superior accuracy compared with other systemic inflammation indexes (Alb, LMR, NLR, PLR, and SII) for the prediction of OS in our study.
CHANG L, et al. [18]	2023	China	62	All male	Median of 60 years	SII of median 598.06 [74.60–3,883.44]	NA	66.13%	Median of 22.61	NA	40	There were no statistically significant diferences between the high and low CONUT score groups in terms of age, performance status (PS), smoking history, alcohol consumption history, BMI, primary tumor site, previous treatment history, pre-RT SII value, and tumor stage (P>0.05).
XU Z, et al. [19]	2023	China	581	M: 410 F: 171	Median of 65 years	NLR: 2 LMR: 3 PLR: 160	Until 2021	5-year OS was 0.629 (95% CI: 0.578–0.676	NA	NA	NA	The results demonstrated markedly better discrimination ability of the nomogram model in terms of C-index.

Table 2 (continued)

Author	Year	Country	Population	Gender (M/F)	Meanage	DII/SII/SIS	Follow-upperiod	Overallsurvival	BMI	Education years	Alcoholuse	Findings
DONG J, et al. [20]	2023	USA	3967	M: 1993 F: 1974	T1: 54.86 ± 7.77 T2: 56.37 ± 7.94 T3: 56.24 ± 7.84	DII T1: (−3.694–1.994) T2: (1.995–2.944) T3: (2.945–5.474)	2 years	NA	Over 170000 under 23.9	Primary school and lower: T1 794, T2 920, T3 935. Junior school: T1 462, T2 372, T3 339. High school and above: T1 66, T2 31, T3 48.	1479	positive though non-significant association between DII scores and ESCC.
LI S, et al. [21]	2022	China	1064 (Case: 532 Control *p*: 532)	M: 373 F: 159 of cases	60 years	DII case 3.05 [2.25;3.89] control 2.38 [1.50;3.43]	NA	NA	NA	Illiteracy/Primary school 332, Junior high school/above 200 of controls.	300	Higher DII in cases

bias ($P < 0.05$), a trim and fill analysis was used to estimate the potential missing effect sizes and to determine a revised overall effect [22].

Sensitivity analysis

Additionally, a sensitivity analysis was carried out on the results of the meta-analysis using the one-study-removed method to evaluate the impact of a specific study on the overall estimation of effects [23].

Results

Study selection

As mentioned earlier, our comprehensive and systematic search in the four databases yielded 67 records. We excluded 49 articles, with 23 of them due to being duplicates by screening the titles and abstracts. Seven of the remaining records were excluded due to the unavailability of the full text. five further articles were then disqualified during the full-text screening process. Finally, our review comprised 13 articles. Six publications provided sufficient data for a meta-analysis (Fig. 1) [9–21].

Study characteristics

Publications included in this review were carried out between 2015 and 2023. The majority of studies took place in China ($n = 9$), whereas a minority of studies were conducted in the USA ($n = 2$), Italy ($n = 1$), and Japan ($n = 1$). The total number of people studied in the included records reached 10,150. The minimum average age of the population studied in these publications was 59.5 years, and it was conducted in China in 2018. The maximum was a median of 70 years, carried out again in China. Six studies were also included for a meta-analysis with sufficient data on DII. We also checked for other inflammatory indexes/scores for our systematic review, with five on SII (systematic inflammatory index) and one on SIS (systematic inflammatory score) (supplementary Table 1). The Food Frequency Questionnaire (FFQ) was utilized in the research to assess the dietary intake status, and the Shivapa method was applied to calculate DII based on 23–36 dietary components [24].

Findings

In the systematic part of the study, all the studies with data on inflammatory indexes/scores demonstrated a clear correlation between an increase in the index/score and cancer incidence. In 9 of the included studies, alcohol drinking was represented as a subgroup where there was a significantly higher prevalence of ESCC among the users.

A total of 10,150 participants were involved in this meta-analysis. The participants were divided into two groups based on their assigned DII: lowest DII and highest DII. Low DII was assumed as a control group that made up 3203 participants. The high DII group was comprised of 6947 individuals. The mean age of the case and control group was calculated as 49.7 ± 5.45 years and 50.6 ± 6.01 years, respectively.

Random effect model implementation on the OR analysis of participants revealed an odds ratio of 1.29 (95%CI: 1.16,1.43, $I^2 > 50\%$) (Figs. 2 and 3). This meant that the high DII group was 29% more likely to develop esophageal cancer compared to the low DII group.

Egger's test for publication bias indicated the existence of bias ($p < 0.050$) (Fig. 4).

Subgroup analysis

The subgroup analysis was conducted to explore potential sources of heterogeneity and examine the association between the Dietary Inflammatory Index (DII) and esophageal cancer risk across different population characteristics. Participants were categorized into low DII (control) and high DII (case) groups, comprising 3203and 6947 individuals, respectively. A random-effects model was applied to assess the pooled odds ratio (OR), revealing that individuals in the high DII group had a 29% increased likelihood of developing esophageal cancer compared to those in the low DII group (OR: 1.29; 95% CI: 1.16–1.43; $I^2 > 50\%$).

Subgroup analysis by geographic location showed that studies conducted in China ($n = 9$) demonstrated a stronger association between pro-inflammatory diets and esophageal cancer risk, while studies from the USA, Italy, and Japan exhibited a slightly lower effect size. Additionally, age stratification indicated that the mean age of participants in both case and control groups was 49.7 ± 5.45 years and 50.6 ± 6.01 years, respectively, with older individuals exhibiting a higher DII-related cancer risk.

A notable finding was observed in alcohol consumption, where nine studies provided data for a subgroup analysis. Results indicated that alcohol users had a significantly higher prevalence of esophageal squamous cell carcinoma (ESCC), supporting the role of both dietary inflammation and lifestyle factors in cancer progression. Furthermore, subgroup analysis based on education level revealed that individuals with lower educational attainment tended to have higher DII scores, though variations in study methodologies prevented precise effect estimation.

Beyond DII, other inflammatory markers were evaluated, including Systemic Inflammatory Index (SII) and Systemic Inflammatory Score (SIS). Studies assessing these markers demonstrated a consistent positive association between increased inflammatory scores and esophageal cancer incidence, further reinforcing the impact of systemic inflammation on cancer risk.

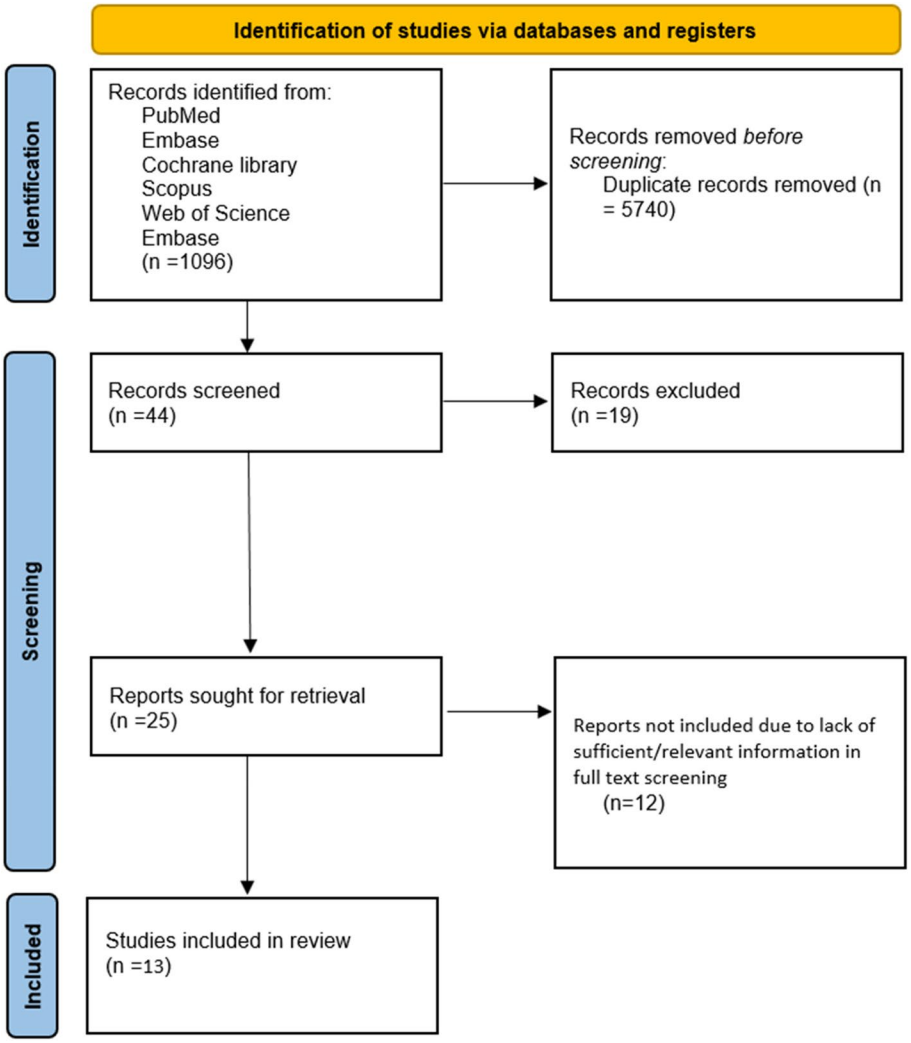

Fig. 1 PRISMA flow chart of the study selection procedure

Sensitivity analysis

To assess the robustness of the findings, a one-study-removed sensitivity analysis was conducted. This method systematically removed one study at a time to determine its impact on the overall pooled effect. The results remained statistically significant across all iterations, with minimal changes in the effect size, confirming that no single study disproportionately influenced the overall association.

the subgroup and sensitivity analyses confirmed that a pro-inflammatory diet, as measured by DII, is significantly associated with an increased risk of esophageal cancer, with lifestyle factors such as alcohol consumption and lower education levels further exacerbating this risk. These findings highlight the importance of dietary inflammation in cancer prevention and the need for dietary modifications to mitigate esophageal cancer risk.

Discussion

Esophageal cancer remains one of the most lethal malignancies globally, despite advancements in diagnostic techniques, therapeutic regimens, and supportive care. Traditional risk factors—such as smoking, alcohol consumption, and low-fiber diets—account for the majority of esophageal squamous cell carcinoma cases in regions like the United States [25]. For esophageal adenocarcinoma, Barrett's esophagus often emerges as a key precursor condition, sharing many of the same environmental and dietary risk factors [26]. Underlying these diverse clinical entities is a growing recognition that chronic, low-grade inflammation can significantly contribute to tumor initiation, progression, and metastasis.

Against this backdrop, our systematic review and meta-analysis provide an updated examination of the

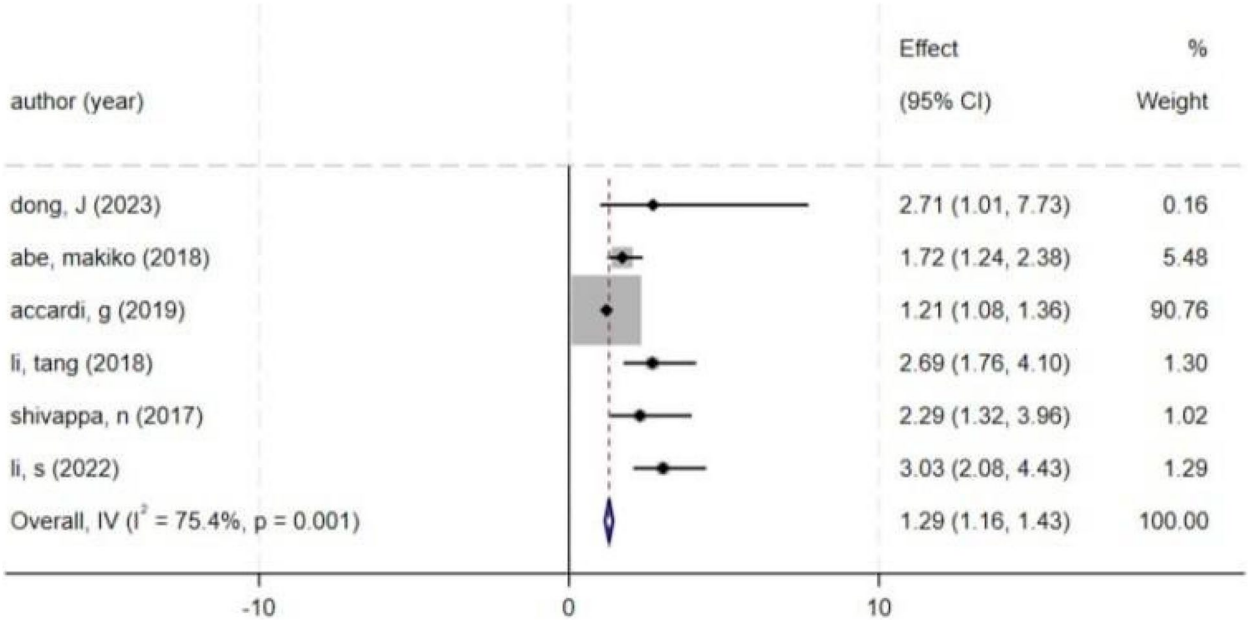

Fig. 2 Forest plot of OR demonstration of esophageal cancer between low DII and high DII groups

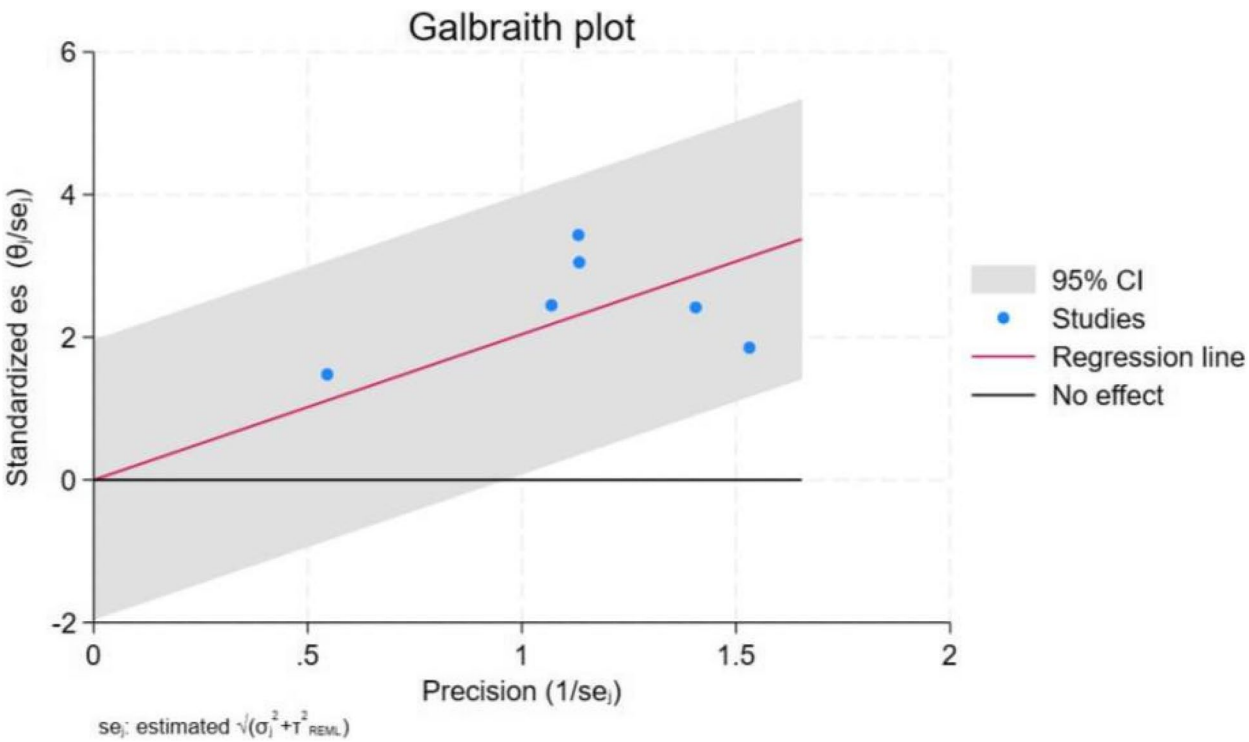

Fig. 3 Galbraith plot for heterogeneity demonstration ($I^2 > 50\%$)

association between dietary inflammatory load, as measured by the Dietary Inflammatory Index (DII), and esophageal cancer risk. The finding that individuals consuming highly inflammatory diets were 29% more likely to develop esophageal cancer than those consuming minimally inflammatory diets underscores the potential importance of dietary patterns in shaping cancer risk. This finding is broadly consistent with

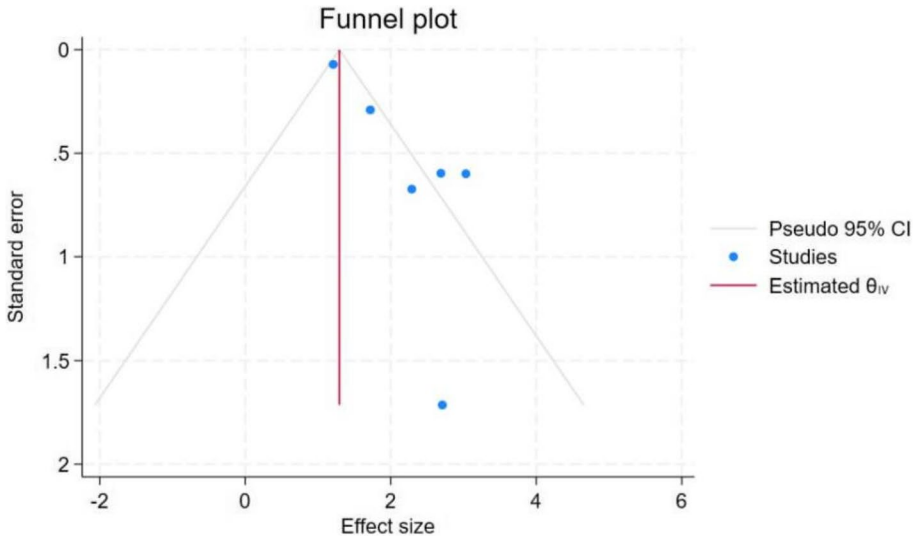

Fig. 4 Funnel plot demonstrating publication bias via asymmetric properties

prior research linking pro-inflammatory diets to various malignancies [27]. Notably, a significant proportion of the studies included in this meta-analysis were conducted in China among older adults [28]. While this demographic focus reveals the relevance of DII in one specific population, it may also constrain the broader applicability of results to other age groups, dietary patterns, and ethnic backgrounds.

Beyond DII, the review also highlighted the role of other systemic inflammatory measures, such as the Systemic Inflammatory Index (SII) and the Systemic Inflammatory Score (SIS), in the risk stratification and prognostication of esophageal cancer [29–35]. These complementary markers may reflect overlapping pathways of chronic inflammation, driven by both dietary and intrinsic host factors. By continuing to investigate these markers in tandem, future research may clarify which approaches best capture the multifaceted nature of cancer-related inflammation and refine clinical decision-making for high-risk individuals.

Strengths

One strength of this investigation is its comprehensive methodological approach. The systematic search spanned multiple databases (PubMed, Web of Science, Scopus, Cochrane library and Embase), and a manual reference search was performed to minimize the risk of missing relevant studies. Moreover, the rigorous meta-analytic techniques—including the use of a random-effects model and Egger's test for publication bias—enhance the reliability of the pooled estimates. Additionally, by exploring multiple inflammatory indexes (DII, SII, SIS), this review offers a broader perspective on how both diet-specific and systemic measures of inflammation may influence esophageal carcinogenesis.

Limitations

Despite these strengths, several limitations warrant caution in interpreting the results. First, geographical and demographic constraints are evident: the majority of participants were from China, limiting generalizability to other populations with distinct genetic backgrounds and dietary habits. Second, many of the included articles used observational designs (case–control or cross-sectional), raising potential issues of recall bias and confounding—particularly for lifestyle factors such as smoking status, physical activity, and body mass index.

A significant source of heterogeneity in our analysis stems from the variability in DII calculation methods, differences in dietary assessment tools (such as Food Frequency Questionnaires), and variations in the number and types of dietary components included across studies. These methodological discrepancies, combined with diverse population characteristics (e.g., age, ethnicity, and regional dietary patterns), contribute to inconsistent DII estimates and may partly explain the observed heterogeneity in effect sizes.

In addition, several studies have demonstrated that higher DII scores correlate with elevated levels of systemic inflammatory markers and cytokines—including TNF-α and IL-6—which reinforces the biological plausibility that a pro-inflammatory diet can drive chronic inflammation. These biomarkers serve as intermediary signals that link dietary exposures with the molecular pathways involved in carcinogenesis, thereby validating the DII as a useful proxy for inflammation.

Diet plays a critical role in modulating inflammation, with specific dietary patterns either exacerbating or mitigating inflammatory responses. Diets rich in refined carbohydrates, saturated fats, and processed foods tend to increase the production of pro-inflammatory mediators and reactive oxygen species, whereas diets high in fruits, vegetables, whole grains, and omega-3 fatty acids promote anti-inflammatory effects. The presence of antioxidants, fiber, and polyphenols in these healthier diets helps reduce oxidative stress and supports a balanced gut microbiome, collectively lowering the overall inflammatory burden.

Third, although the DII was calculated using standard protocols (e.g., the Shivappa method), variations in the number and type of dietary components included may introduce measurement inconsistencies across studies. Finally, publication bias was detected by Egger's test, implying that smaller null-result studies might be underrepresented, potentially inflating the effect size reported here.

Taken together, these findings underscore the integral role of inflammation—both systemic and diet-induced—in esophageal oncogenesis. Future research should strive for greater diversity in study populations, employ longitudinal cohort designs, and systematically address potential confounding variables to solidify our understanding of how dietary inflammation influences cancer risk. In particular, standardizing DII measurement protocols will facilitate meaningful cross-study comparisons. Such efforts may ultimately yield targeted dietary guidelines and interventions to curb inflammation and reduce the global burden of esophageal cancer.

Conclusion

This systematic review and meta-analysis provide compelling evidence that a higher Dietary Inflammatory Index (DII) score significantly increases the likelihood of developing esophageal cancer. Across the included studies, individuals with elevated DII scores were 29% more susceptible to this malignancy compared with those consuming less inflammatory diets. Moreover, other systemic inflammatory markers—such as SII and SIS—also showed consistent associations with disease progression and prognosis, underscoring the broader impact of diet-induced inflammation in carcinogenesis. While most data were derived from older Chinese populations, the findings highlight a global need for further investigation into how anti-inflammatory dietary patterns may reduce esophageal cancer risk in diverse racial and ethnic groups. Ultimately, integrating dietary intervention strategies and monitoring inflammatory biomarkers could become an integral part of risk assessment, prevention, and patient management for esophageal cancer.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12885-025-14199-5.

Supplementary Material 1.

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Authors' contributions
Study concept and design: MAA acquisition of the data: YK,SASM Analysis and interpretation of the data: AE,MB,KS,AS Drafting of the manuscript: OR.; critical revision of the manuscript for important intellectual content: HM,MA,HBY,NR,HKN,FK,AM administrative, technical, and material support: ZM,SKSR study supervision: MAA.

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Data availability
Data is provided within the manuscript or supplementary information files.

Declarations

Ethical approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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