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Meta-analysis of the relationship between Dietary Inflammatory Index and esophageal cancer risk

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Abstract

Introduction: Diet is closely related to the occurrence of esophageal cancer (EC). Dietary Inflammatory Index (DII), as a novel index that describes the inflammatory potential of diet, was widely used in many diseases.

Objective: To systematically analyze the relationship between DII and the risk of esophageal cancer.

Methods: We mainly searched relative studies in PubMed, Cochrane library, Web of Science, and other literature database. The random-effect model was used for meta-analysis, and subgroup analysis and sensitivity analysis were used to detect the origin of heterogeneity.

Results: We finally obtained 6 articles (8 studies). All studies were case-control studies which consisted of 1961 cases and 3577 controls. In this study, compared with the lowest DII category, the highest DII category had a higher risk of esophageal cancer, and the pooled odds ratio (OR) of the 8 studies were 2.54 (95% confidence interval (CI): 1.90–3.40; $I^2 = 65.7\%$, $P = .005$). Furthermore, regardless of the differences in published year, DII components, geographic location, and study quality, there was still an increased risk of esophageal cancer in the highest DII category compared with the lowest DII category.

Conclusions: Our results inferred that DII was positively correlated with esophageal cancer risk and it could be used as a tool to predict the esophageal cancer risk and evaluate human health.

Abbreviations: DALYs = disability-adjusted life years, DII = dietary Inflammatory Index, EC = esophageal cancer, FFQ = Food Frequency Questionnaire.

Keywords: Dietary Inflammatory Index, esophageal cancer, inflammation, meta-analysis

1. Introduction

Esophageal cancer (EC), as the ninth most common cancer in the world, is a fatal disease. World Health Organization reported that 572,034 people developed esophageal cancer and 508,585 people died of EC in 2018.[1] And about one third of disability-adjusted life years (DALYs) due to cancer were caused by EC,

studies reported.[2,3] These indicate that the incidence status, mortality status, and disease burden of EC are still very severe. Therefore, it is imperative to constantly study the etiology and find out the inducing factors of EC, and take this as a breakthrough point for prevention and treatment of EC.

To our knowledge, esophageal cancer is a disease caused by many factors. Except for genetic factors, environmental factors, nutrition and exercise factors, dietary factors are closely related to it.[4] A scientific and reasonable diet is helpful to prevent the occurrence of esophageal cancer. Studies have shown that many food components can produce many bioactive substances related to chronic inflammation in the body.[5–8] In recent years, an increasing number of studies have demonstrated that chronic inflammation plays an essential medium in the occurrence and development of tumor.[9–12] We can use this as a pointcut to study the relationship between dietary factors and the esophageal cancer risk.

Dietary Inflammatory Index (DII), developed by Shivappa in 2014, could objectively describe the diet inflammatory potential, according to a large number of literatures, population data and the anti-inflammatory and pro-inflammatory effects of various dietary factors on 6 serum inflammatory markers.[13] There are a total of 45 food components calculated for DII, including 9 pro-inflammatory components and 36 anti-inflammatory components.[13] The calculation of DII needs to obtain dietary information from the Food Frequency Questionnaire (FFQ), to calculate the content of various food ingredients, and then calculate individual DII by specific formula according to the index of 45 Food ingredients. In a population, inflammatory potential of diet increased with the increase of DII score. Studies have identified that DII score is positively correlated with several

inflammatory factors, including TNF-α, CRP, homocysteine, and IL-6.[14–17] Other studies have also indicated that serum inflammatory cytokines such as CRP and IL-6 are closely associated with several tumors risk.[18–21] So we hypothesized that DII could be used to monitor dietary inflammation and health status.

Increasingly, researches were conducted on the connection between DII and esophageal cancer. And the relationship between DII and EC has not been reviewed. Therefore, our purpose is to conduct a systematic review on the relationship between DII score and esophageal cancer risk, to explore whether the DII could be used as a tool to predict the occurrence and development of esophageal cancer, and to determine the health status of people. It can provide theoretical basis for evaluating the status of dietary inflammation and predicting the occurrence and development of esophageal cancer.

2. Methods

2.1. Search strategy

The relative studies were searched mainly in PubMed, Cochrane library, Web of Science and other literature database. The main search strategy terms were as follows: (((dietary inflammatory index OR inflammatory diet OR the potential inflammatory of diet OR anti-inflammatory diet OR dietary score) AND (esophagus OR oesophagus)) AND (cancer OR carcinoma OR neoplasm OR tumor)). In addition, when we reviewed abstracts and full texts of relevant studies in the database, we searched for similar articles recommended by the database and the references of relevant studies. There were no restrictions on country and type of design, but only English articles published after 2014 (DII was proposed in 2014) were considered.

2.2. Inclusion and exclusion criteria

Inclusion criteria:

1. The literatures are retrospective studies or prospective studies.
2. The DII in the literature is measured by the original data.

   Exclusion criteria:

1. The full text of the literature cannot be obtained.
2. Literature is a review.
3. The original text does not contain the descriptions of DII and esophageal cancer.

   Studies that meet both of these inclusion criteria will be included. However, if the study met one of the exclusion criteria, they would be excluded.

2.3. Quality evaluation of included articles

Two authors (Liang Ou and Kai Li) reviewed the quality of selected articles by using Newcastle-Ottawa Scale (NOS) tool,[22] including selection, comparability, and outcome assessment. Studies with a NOS score above 7 were considered as high-quality studies.

2.4. Data extraction

The information of included articles were extracted as follows: First author, publication year, country, sample of size, sex, age, Food Frequency Questionnaire (FFQ) items, DII components, adjustments, Odds ratios (ORs), 95% confidence intervals (CIs). In this study, we extracted the multivariate adjusted ORs and 95%CIs. For different ORs and 95%CIs levels, we only extracted the ORs and 95%CIs of the comparison between the highest and the lowest category of DII. When there were no overall ORs and 95%CIs in the article, only the classified ones could be obtained, then the ORs and 95%CIs of each related category were extracted respectively.

2.5. Ethical approval

Because this study is a meta-analysis and the data are based on previously published literature, ethical approval is not necessary.

2.6. Statistical analysis

The ORs and 95% CIs were combined by meta-analysis to evaluate the relationship between DII and risk of EC. The results of meta-analysis were presented by forest plot. Because DII score was used as continuous variables or categorical variables in studies and only 2 studies included continuous variables, we mainly performed statistical analysis on categorical variables. For articles containing multiple categories, the relevant categories were analyzed as independent studies. In this study, Cochranes Q test was used to identify the heterogeneity, $P < .1$ was regarded as significant heterogeneity. Inconsistency (I^2) was used to evaluate the size of heterogeneity. $I^2 \leq 50\%$ indicates low heterogeneity, using fixed-effect model. $50\% < I^2 \leq 75\%$ suggests significant heterogeneity, utilizing random-effect model.[22] And we had better to find out the sources of heterogeneity. $I^2 > 75\%$ meant that there was considerable heterogeneity among the studies, and it was rather necessary to explore and analyze the origins of heterogeneity.

To explore the origin of heterogeneity, subgroup analyses were carried out on published year, DII components, geographical location and study quality of relevant researches. In addition, sensitivity analysis was performed by removing the studies one by one, and new heterogeneity should be recorded when the studies were removed. Egger or Begger test should be used to detect the publication bias when ten or more researches were included, and $P > .05$ could be considered as no publication bias.[23]

All of the statistical analyses and results display of this study were performed using STATA version 11.0.

3. Results

3.1. Study search and characteristics

In this study, 126 documents were retrieved according to the retrieval strategy. Among that, 34 articles were retrieved from PubMed, 39 articles from Cochrane Library, 23 articles from Web of Science, 30 articles from other databases. Twenty two duplicate articles were eliminated and 91 articles were excluded based on the title and abstract of literatures. Then, 13 literatures were obtained for full-text evaluation. Other 7 literatures unmatched the inclusion criteria were excluded (4 were reviews, 3 did not contain the description of esophageal cancer). Finally, 6 articles (8 studies) were obtained, all of which were case-control studies.[16,24–28] The detailed process was displayed in Figure 1.

The included articles were published between 2015 and 2018, 8 case-control studies had 1961 cases and 3577 controls (Males:
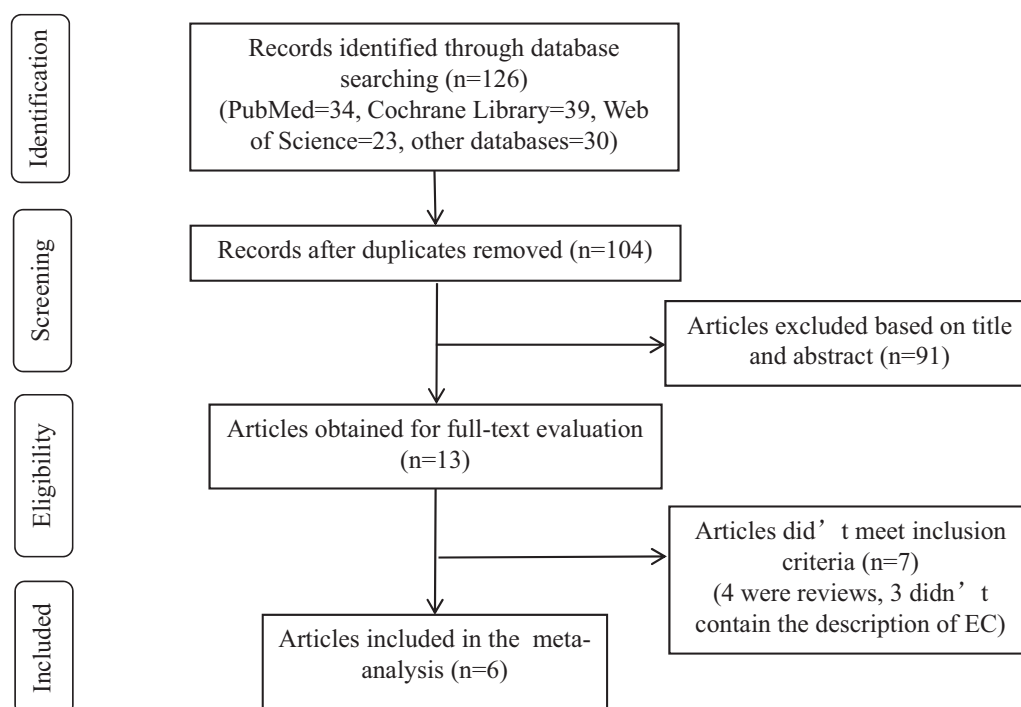
**Figure 1.** Flow chart of the literature screening and selection process for the meta-analysis. It shows the screening and inclusion process of the study. EC = esophageal cancer.

Table 1

Characteristics of 6 articles in the meta-analysis.

First author [references], year, country	Study design	Cases, n	Total participants, n	Age	DII components (FFQ items)	OR (95%CI)	Adjustments	NOS score
Shivappa,[24] 2015, Italy	Case-control	304	1047	39–77	31 (78)	Overall: High vs low: 2.46 (1.40–4.36); Continuous: 1.39 (1.25–1.54)	Age, Sex, Year of interview, Area of residence, Alcohol intake, Smoking status, BMI, Physical activity, Aspirin use	7
Shivappa,[25] 2015, Iran	Case-control	47	143	40–75	27 (125)	Overall: High vs low: 8.24 (2.03–33.47); Continuous: 3.58 (1.76–7.26)	Age, Sex, Education, Physical activity, BMI, Smoking status, Gastroesophageal reflux	7
Lu,[26] 2015, Sweden	Case-control	594	1155	19–80	36 (63)	Oesophageal squamous cell cancer: High vs low: 4.35 (2.24–8.43); Oesophageal adenocarcinoma: High vs low: 3.59 (1.87–6.89); Gastroesophageal junctional adenocarcinoma: High vs low: 2.04 (1.24–3.36)	Age, Sex, Total energy intake, Education, Smoking status, Alcohol intake, Physical activity, Gastroesophageal reflux, Helicobacter pylori infection	6
Shivappa,[27] 2017, USA	Case-control	224	405	64.3±11.2	25 (101)	Overall: High vs low: 2.29 (1.32–3.96)	Age, Sex, Total energy intake, Smoking status, BMI, Occupation, Alcohol intake, NSAIDs use, Helicobacter pylori infection	8
Tang,[28] 2018, China	Case-control	359	739	61.0±11.4	23 (137)	Overall: High vs low: 2.55 (1.61–4.06)	Age, Sex, Education, BMI, Total energy intake, Smoking status, Alcohol intake, Family history of cancer in first-degree relatives	7
Abe,[29] 2018, Japan	Case-control	433	1729	60±10.9	23(47)	Overall: High vs low: 1.71 (1.54–1.90)	Smoking status, Alcohol intake, flushing, phenotype, Number of teeth, occupation	8

OR = odds ratio, CI = confidence interval, DII = Dietary Inflammatory Index, FFQ = Food Frequency Questionnaire, High vs low = the highest DII category compared with the lowest DII category, Continuous = DII is used as a continuous variable, NOS = Newcastle-Ottawa Scale.
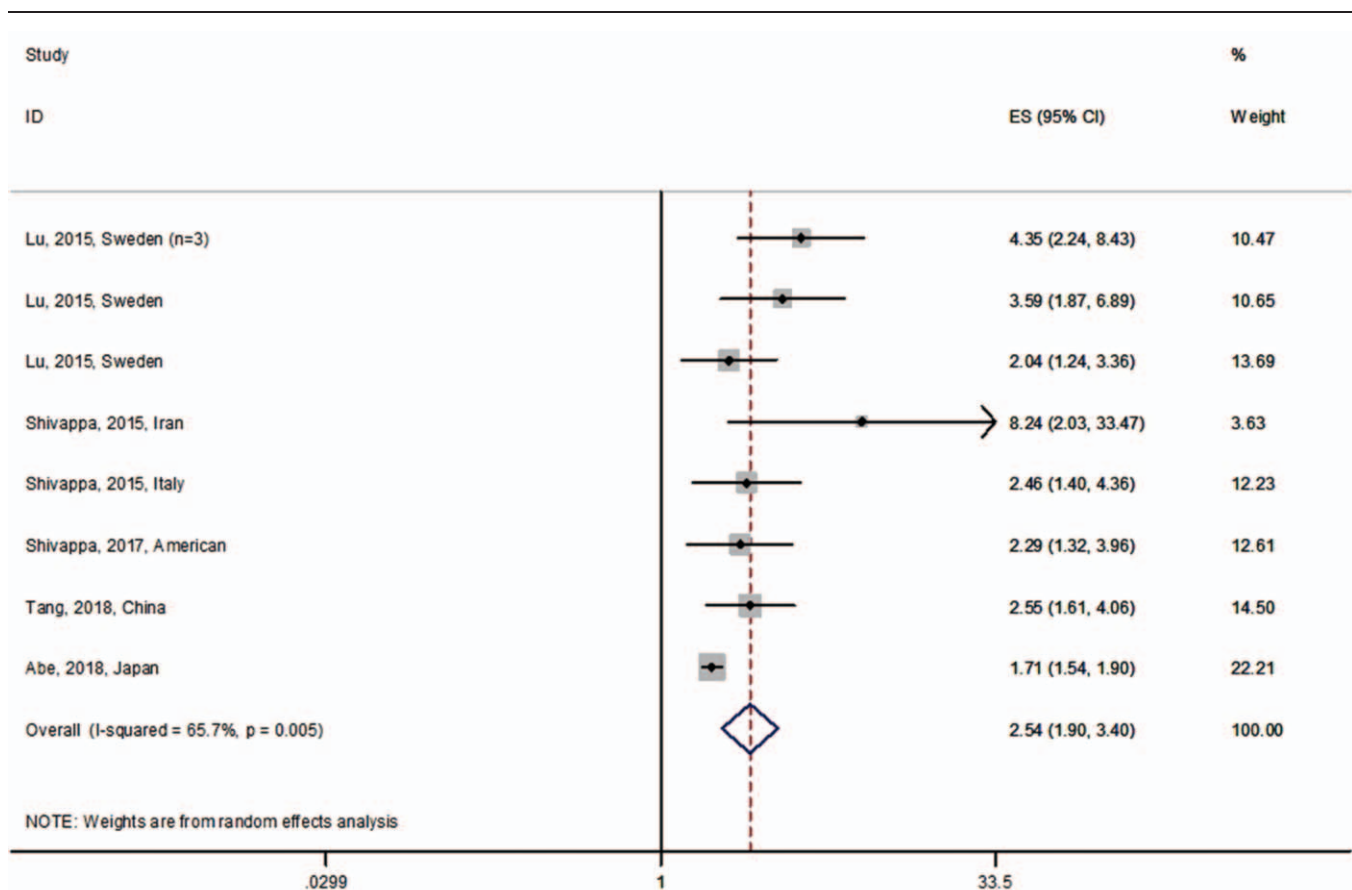
Figure 2. A forest plot (Summary estimates for the odds ratios (ORs) of the highest compared with the lowest catagory of dietary inflammation index (DII) and esophageal cancer). It indicates that the highest DII group had an increased risk of esophageal cancer compared to the lowest group. OR = odds ratio, CI = confidence interval.

4511, Females: 1027). All of the studies used Food Frequency Questionnaire (FFQ) to evaluate the dietary intake status, and 23 to 36 components of diet were used to calculate DII by using the method of Shivapa.[13] The 6 articles were from 6 countries, including 1 from Iran, 1 from Italy, 1 from Sweden, 1 from USA, 1 from China and 1 from Japan. The detailed characteristics of relevant articles were shown in Table 1.

3.2. Quality assessment

The NOS score of 6 literatures ranged from 6 to 8 points, among which 5 articles with the NOS score ≥7 points were of high quality. The detailed NOS score of all relevant articles is shown in Table 1.

3.3. DII and esophageal cancer risk

As shown in the forest blogs (Fig. 2). The risk of EC in the highest category of DII was 2.54 times higher than that in the lowest. The pooled OR of 8 studies was 2.54 (95%CI: 1.90–3.40; $I^2 = 65.7\%$, $P = .005$; Fig. 2).

3.4. Subgroup analysis and sensitivity analysis

Subgroup analyses were carried out to detect the sources of the heterogeneity in this study ($I^2 = 65.7\%$, $P = .005$). Subgroup analysis of published year showed significant heterogeneity in studies published in 2018 ($I^2 = 63.3\%$, $P = .099$) and no significant heterogeneity in studies published in 2015 ($I^2 = 35.7\%$, $P = .183$). There were significant heterogeneity in studies with <30 DII components ($I^2 = 63.8\%$, $P = .041$) and no significant heterogeneity in studies with ≥30 DII components ($I^2 = 25.1\%$, $P = .261$). Studies in Asia showed significant heterogeneity ($I^2 = 63.8\%$, $P = .041$), while studies in Europe showed no significant heterogeneity ($I^2 = 25.1\%$, $P = .261$). There were significant heterogeneity in studies of NOS score ≥7 ($I^2 = 58.0\%$, $P = .049$) and no significant heterogeneity in studies of NOS score <7 ($I^2 = 46.9\%$, $P = .0.152$). The detailed results of subgroup analysis were shown in Table 2. The results of subgroup analyses implied that the differences in published year, DII components, geographic location, study quality were not the source of heterogeneity in this study.

Sensitivity analysis was carried out to further identify the source of heterogeneity. We removed relevant studies one by one and recorded changes in heterogeneity. When Abes study[28] was removed, the heterogeneity reduced ($I^2 = 12.8\%$, $P = .332$), while the heterogeneity did not change much when other studies were removed. The heterogeneity of meta-analysis after removal of a certain study is shown in Table 3. In the multivariate adjustment analysis, Abes study[28] failed to include age and gender into the multivariate adjustment model and only 6 variables were included into the multivariate adjustment model. In addition,

Table 2

the results of subgroup analysis (published year, DII components, geographic location, study quality).

Subgroup	Studies, n	Model	Pooled OR (95% CI)	Heterogeneity	
				I^2 (%)	P value
All studies	8	Random	2.54 (1.90–3.40)	65.7	.005
Published year					
2015	5	Fixed	3.06 (2.11–4.43)	35.7	.183
2017	1	Fixed	2.29 (1.32–3.97)	NA	NA
2018	2	Random	1.95 (1.35–2.83)	63.3	.099
DII components					
≥30	4	Fixed	2.03 (2.01–3.97)	25.1	.261
<30	4	Random	2.26 (1.54–3.31)	63.8	.041
Geographic location					
Asia	3	Random	2.38(1.39–4.06)	73.0	.024
North America	1	Fixed	2.29(1.32–3.97)	NA	NA
Europe	4	Fixed	2.83(2.01–3.97)	25.1	.261
Study quality					
NOS score ≥7 (high quality)	5	Random	2.25(1.64–3.10)	58.0	.049
NOS score <7	3	Fixed	3.04(1.87–4.88)	46.9	.152

OR = odds ratio, CI = confidence interval, DII = Dietary Inflammatory Index, NA = not available, NOS = Newcastle-Ottawa Scale.

the Food Frequency Questionnaire (FFQ) of the study was only 47 items, which was the least among the 8 studies. And Abes[28] study was a gray literature, considering the result of sensitivity analysis, the study might be the source of heterogeneity in the meta-analysis.

Since less than 10 studies were included, publication bias could not be assessed.

4. Discussion

In this study, meta-analysis was performed on the relationship between DII and esophageal cancer risk in 6 articles (8 studies).[24–28] The results suggested that the risk of esophageal cancer in the highest DII category was 2.54 times higher than that in the lowest (OR: 2.54, 95%CI: 1.90–3.40). Besides, regardless of the differences in published year, DII components, geographic location and study quality, the highest DII group had a higher risk of esophageal cancer than the lowest group. The results of our study are consistent with the meta-analysis on other cancers.[29–32]

Attention should be paid to the heterogeneity of our study. In this study, we conducted subgroup analysis on 4 factors (published year, DII components, geographic location, study quality) of relevant studies. And there was still high heterogeneity in the subgroups. So DII components, published year, geographic

Table 3

The heterogeneity of the meta-analysis after the following studies was removed.

Studies	I^2 (%)	$P_{Heterogeneity}$
Shivappa,[24] 2015, Italy	69.2	.000
Shivappa,[25] 2015, Iran	62.7	.013
Lu,[26] 2015, Sweden, Oesophageal squamous cell cancer	57.1	.030
Lu,[26] 2015, Sweden, Oesophageal adenocarcinoma	63.5	.012
Lu,[26] 2015, Sweden, Gastroesophageal junctional adenocarcinoma	70.4	.002
Shivappa,[27] 2017, USA	69.8	.003
Tang,[28] 2018, China	67.7	.005
Abe,[29] 2018, Japan	12.8	.332

location and study quality were not the sources of heterogeneity in this study. Although there was heterogeneity among subgroups, the risk of esophageal cancer in the highest DII group was still higher than that in the lowest. We carried out sensitive analysis by removing the studies one by one, and the result showed that Abes study might be the source of heterogeneity. And we further studied the contents of Abes article.[28] The results showed that the FFQ items and adjustments of the study were less than other studies, and it was a gray literature. These probably made it different from other studies. Nevertheless, in Abes study, DII was positively correlated with the risk of esophageal cancer.[28] Therefore, we inferred that DII could be used as a tool to predict the risk of esophageal cancer and to evaluate the state of human health.

Dietary Inflammatory Index (DII) is an updated, low-consumption, novel, and readily available index, which can describe the state of dietary inflammation. DII has been shown to be positively correlated with some inflammatory factors such as CRP, TNF-α, IL-6, and homocysteine.[14–16] Rencently, a Japanese research also showed a similar correlation between DII and CRP levels.[17] These inflammatory factors were closely related to the occurrence of chronic inflammation, which could promote the development of tumors.[9–11,33]

A series of bioactive substances produced by diet could induce the occurrence of chronic inflammation in the body.[5–8] Some rational explanations have been proposed for diet-induced inflammation. In the DII system, energy, carbohydrates, and total fats are consider as pro-inflammatory components that could induce system inflammation through increasing body weight.[34] Saturated fatty acids (SFAs) could induce worse inflammation by promoting the activation and proliferation of NF-κB, protein kinase C, mitogen-activated protein kinases, and the induction of inflammatory genes.[35] Trans fatty acids (TFAs) can increase the activation of TNF system[36] and promote the production of CRP, IL-6 and other inflammatory factors, leading to system inflammation[37,38]. Cholesterol promotes inflammation through accumulation in immune cells. And its mechanisms include activation of inflammasome, enhancement of toll-like receptor (TLR) signaling, production of monocytes and neutrophils in bone marrow and spleen[39]. To our knowledge,

saturated fats (SFAs) could be found in animal fats, palm oil, cocoa butter, coconut oil, etc. Trans fatty acids (TFAs) are widely found in margarine, cakes, cookies, fried foods, cheeses, etc. Cholesterol is present in meat, eggs and fish, especially in animal livers and egg yolk. We can reduce the occurrence of chronic inflammation by controlling our weight and decreasing the intake of these pro-inflammatory food components. What should remind especially is, we should take scientific and effective method when controlling weight, cannot be blind.

In the DII system, some possible explanations of some anti-inflammatory food components have also been proposed. Vitamin E and vitamin C could decrease system inflammation through reducing pro-inflammatory cytokine expression and decreasing oxidative stress.[36,40] N-3 fatty acids could increase anti-inflammatory effects by reducing the production of hydrogen peroxide and activation of NF-κB.[41] Zinc, an anti-inflammatory mineral, could decrease activation of NF-κB, TNF-α and IL-1β and increase the gene expression of double zinc finger protein, A20 and PPAR-α, which have anti-inflammatory effects.[42] Polyunsaturated fatty acids (PUFAs) can achieve anti-inflammatory effects through inhibiting the production of TNF-α and NO.[43] To take an example, the Mediterranean diet contains a great many of anti-inflammatory components such as monounsaturated fatty acids (MUFAs), omega-3, omega-6 polyunsaturated fatty acids (PUFAs), many vegetables, beans, and grains rich in various vitamins, which have anti-inflammatory effects on human body.[33,44–46] So we can increase the intake of such food components to make our body more anti-inflammatory.

There are some strengths in the study. This study is the first systematic review on the relationship between DII and esophageal cancer risk. Moreover, DII is used to describe dietary inflammation, which is more comprehensive and objective than a single nutrient. And DII could be used as a tool to screen esophageal cancer and conduct public nutrition interventions in the future. And it has important guiding significance to predict the risk of esophageal cancer and evaluate human health. However, there are several limitations in the study. First, all researches included in this study were case-control studies. DII was extracted from the FFQ based on participants self-reports, which might lead to a degree of recall bias. Second, there was substantial heterogeneity in this study, and it might be caused by the Abes study.[28] It might because Abes study[28] failed to include age and gender into the multivariate adjustment model and only 6 variable were included into the multivariate adjustment model. Besides, FFQ items in the study were the least among all the included studies and the study was a gray literature. Third, there were only 6 articles (8 studies) included in the study. Further research is required to include more studies in the future.

In conclusion, our results suggested that DII was positively correlated with esophageal cancer risk, that is, people with relatively high dietary inflammatory potential were more likely to develop esophageal cancer. Therefore, DII could be used as a tool to predict the risk of esophageal cancer and evaluate human health. We believe that, on the premise of ensuring reasonable nutrition, reducing the intake of pro-inflammatory food ingredients and increasing anti-inflammatory food ingredients have important guiding significance for preventing the occurrence of esophageal cancer.

Author contributions

Data curation: Qiu-Jin Chen.
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Methodology: Qiu-Jin Chen.
Project administration: Fengrong Ou.
Resources: Kai Li.
Supervision: Kai Li.
Validation: Fengrong Ou.
Writing – original draft: Qiu-Jin Chen.
Writing – review & editing: Liang Ou, Fengrong Ou.

References

[1] Bray F, Ferlay J, Soerjomataram I, et al. Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2018;68:394–424.

[2] Fitzmaurice C, Abate D, Abbasi N, et al. Global, regional, and national cancer incidence, mortality, years of life lost, years lived with disability, and disability-adjusted life-years for 29 cancer groups, 1990 to 2017: a systematic analysis for the global burden of disease study. JAMA Oncol 2019;5:1749–68.

[3] Fitzmaurice C, Allen C, Barber RM, et al. Global, regional, and national cancer incidence, mortality, years of life lost, years lived with disability, and disability-adjusted life-years for 32 cancer groups, 1990 to 2015: a systematic analysis for the global burden of disease study. JAMA Oncol 2017;3:524–48.

[4] Xie SH, Lagergren J. Risk factors for oesophageal cancer. Best Pract Res Clin Gastroenterol 2018;36-37:3–8.

[5] Ahluwalia N, Andreeva VA, Kesse-Guyot E, et al. Dietary patterns, inflammation and the metabolic syndrome. Diabetes Metab 2013;39: 99–110.

[6] Zitvogel L, Pietrocola F, Kroemer G. Nutrition, inflammation and cancer. Nat Immunol 2017;18:843–50.

[7] Chassaing B, Van de Wiele T, De Bodt J, et al. Dietary emulsifiers directly alter human microbiota composition and gene expression ex vivo potentiating intestinal inflammation. Gut 2017;66:1414–27.

[8] Riccio P, Rossano R. Diet, gut microbiota, and vitamins D+A in multiple sclerosis. Neurotherapeutics 2018;15:75–91.

[9] Shacter E, Weitzman SA. Chronic inflammation and cancer. Oncology (Williston Park) 2002;16:217–26. 229; discussion 230-212.

[10] Landskron G, De la Fuente M, Thuwajit P, et al. Chronic inflammation and cytokines in the tumor microenvironment. J Immunol Res 2014;2014:149185.

[11] Chai EZ, Siveen KS, Shanmugam MK, et al. Analysis of the intricate relationship between chronic inflammation and cancer. Biochem J 2015;468:1–5.

[12] Greten FR, Grivennikov SI. Inflammation and cancer: triggers, mechanisms, and consequences. Immunity 2019;51:27–41.

[13] Shivappa N, Steck SE, Hurley TG, et al. Designing and developing a literature-derived, population-based dietary inflammatory index. Public Health Nutr 2014;17:1689–96.

[14] Shivappa N, Steck SE, Hurley TG, et al. A population-based dietary inflammatory index predicts levels of C-reactive protein in the Seasonal Variation of Blood Cholesterol Study (SEASONS). Public Health Nutr 2014;17:1825–33.

[15] Shivappa N, Hébert JR, Rietzschel ER, et al. Associations between dietary inflammatory index and inflammatory markers in the Asklepios Study. Br J Nutr 2015;113:665–71.

[16] Tang L, Shivappa N, Hebert JR, et al. Dietary inflammatory index and risk of oesophageal cancer in Xinjiang Uyghur Autonomous Region, China. Br J Nutr 2018;119:1068–75.

[17] Yang Y, Hozawa A, Kogure M, et al. Dietary inflammatory index positively associated with high-sensitivity c-reactive protein level in Japanese From NIPPON DATA 2010. J Epidemiol 2020; 30:98–107.

[18] Ose J, Schock H, Tjonneland A, et al. Inflammatory markers and risk of epithelial ovarian cancer by tumor subtypes: the EPIC cohort. Cancer Epidemiol Biomarkers Prev 2015;24:951–61.

[19] Zeng F, Wei H, Yeoh E, et al. Inflammatory markers of CRP, IL 6, TNFalpha, and soluble TNFR2 and the risk of ovarian cancer: a meta-analysis of prospective studies. Cancer Epidemiol Biomarkers Prev 2016;25:1231–9.

[20] Li Y, Zhong X, Cheng G, et al. Hs-CRP and all-cause, cardiovascular, and cancer mortality risk: a meta-analysis. Atherosclerosis 2017;259: 75–82.

[21] Dolan RD, McSorley ST, Horgan PG, et al. The role of the systemic inflammatory response in predicting outcomes in patients with advanced inoperable cancer: systematic review and meta-analysis. Crit Rev Oncol Hematol 2017;116:134–46.

[22] Higgins JP, Thompson SG, Deeks JJ, et al. Measuring inconsistency in meta-analyses. BMJ 2003;327:557–60.

[23] Begg CB, Mazumdar M. Operating characteristics of a rank correlation test for publication bias. Biometrics 1994;50:1088–101.

[24] Shivappa N, Zucchetto A, Serraino D, et al. Dietary inflammatory index and risk of esophageal squamous cell cancer in a case-control study from Italy. Cancer Causes Control 2015;26:1439–47.

[25] Shivappa N, Hebert JR, Rashidkhani B. Dietary inflammatory index and risk of esophageal squamous cell cancer in a case-control study from Iran. Nutr Cancer 2015;67:1253–9.

[26] Lu Y, Shivappa N, Lin Y, et al. Diet-related inflammation and oesophageal cancer by histological type: a nationwide case-control study in Sweden. Eur J Nutr 2016;55:1683–94.

[27] Shivappa N, Hebert JR, Anderson LA, et al. Dietary inflammatory index and risk of reflux oesophagitis, Barrett's oesophagus and oesophageal adenocarcinoma: a population-based case-control study. Br J Nutr 2017;117:1323–31.

[28] Abe M, Shivappa N, Ito H, et al. Dietary inflammatory index and risk of upper aerodigestive tract cancer in Japanese adults. Oncotarget 2018;9:24028–40.

[29] Shivappa N, Godos J, Hebert JR, et al. Dietary inflammatory index and colorectal cancer risk-a meta-analysis. Nutrients 2017;9:1043.

[30] Lu DL, Ren ZJ, Zhang Q, et al. Meta-analysis of the association between the inflammatory potential of diet and urologic cancer risk. PLoS One 2018;13:e0204845.

[31] Zhu Y, Li Q, Xu X. Dietary inflammatory index and the risk of prostate cancer: a dose-response meta-analysis. Eur J Clin Nutr 2019;74:1001–8.

[32] Liu ZY, Gao XP, Zhu S, et al. Dietary inflammatory index and risk of gynecological cancers: a systematic review and meta-analysis of observational studies. J Gynecol Oncol 2019;30:e23.

[33] Coussens LM, Werb Z. Inflammation and cancer. Nature 2002;420:860–7.

[34] Gregor MF, Hotamisligil GS. Inflammatory mechanisms in obesity. Annu Rev Immunol 2011;29:415–45.

[35] Kennedy A, Martinez K, Chuang CC, et al. Saturated fatty acid-mediated inflammation and insulin resistance in adipose tissue: mechanisms of action and implications. J Nutr 2009;139:1–4.

[36] Chien CT, Chang WT, Chen HW, et al. Ascorbate supplement reduces oxidative stress in dyslipidemic patients undergoing apheresis. Arterioscler Thromb Vasc Biol 2004;24:1111–7.

[37] Mazidi M, Gao HK, Kengne AP. Inflammatory markers are positively associated with serum trans-fatty acids in an adult american population. J Nutr Metab 2017;2017:3848201.

[38] Mozaffarian D, Pischon T, Hankinson SE, et al. Dietary intake of trans fatty acids and systemic inflammation in women. Am J Clin Nutr 2004;79:606–12.

[39] Tall AR, Yvan-Charvet L. Cholesterol, inflammation and innate immunity. Nat Rev Immunol 2015;15:104–16.

[40] Murphy RT, Foley JB, Tome MT, et al. Vitamin E modulation of C-reactive protein in smokers with acute coronary syndromes. Free Radic Biol Med 2004;36:959–65.

[41] Lopez-Garcia E, Schulze MB, Manson JE, et al. Consumption of (n-3) fatty acids is related to plasma biomarkers of inflammation and endothelial activation in women. J Nutr 2004;134:1806–11.

[42] Jarosz M, Olbert M, Wyszogrodzka G, et al. Antioxidant and anti-inflammatory effects of zinc. Zinc-dependent NF-κB signaling. Inflammopharmacology 2017;25:11–24.

[43] Ahmad TB, Rudd D, Kotiw M, et al. Correlation between fatty acid profile and anti-inflammatory activity in common australian seafood by-products. Mar Drugs 2019;17:155.

[44] Galland L. Diet and inflammation. Nutr Clin Pract 2010;25:634–40.

[45] Di Lorenzo C, Dell'Agli M, Badea M, et al. Plant food supplements with anti-inflammatory properties: a systematic review (II). Crit Rev Food Sci Nutr 2013;53:507–16.

[46] Zhu F, Du B, Xu B. Anti-inflammatory effects of phytochemicals from fruits, vegetables, and food legumes: a review. Crit Rev Food Sci Nutr 2018;58:1260–70.