Review Article

A Higher Dietary Inflammatory Index Score is Associated with a Higher Risk of Incidence and Mortality of Cancer: A Comprehensive Systematic Review and Meta-Analysis

Abstract

Background: Inflamation is widely known as an adaptive pathophysiological response in a variety of cancers. There is an expanding body of research on the key role of diet in inflammation, a risk factor for all types of cancer. Dietary inflammatory index (DII) was recently develpoed to evalute the inflammatory potential of a diet either as anti-inflammatory or pro-inflammatory. In fact, several studies have shown the association of DII and risk of different cancer types. The aim of this meta-analysis was to investigate the association of DII with risk of incidence and mortality of any cancer types. **Methods:** We searched PubMed-Medline, Scopus, and Web of Science databases for pertient studies util January, 2017. All studies conducted to investigate the association of DII and incidence, mortality, and hospitalization of all cancer types were included. According to degree of heterogeneity, fixed- or random-effect model was employed by STATA software. **Results:** Total 38 studies were eligible for the meta-analysis. The results show that a higher level of DII increases the risk for all cancer types incidence by 32% (OR: 1.32; 95% CI: 1.22-1.42) including digestive tract cancers (OR: 1.55; 95% CI: 1.33-1.78), hormone-dependent cancers (OR: 1.14; 95% CI: 1.04-1.24), respiratory tract cancers (OR: 1.64; 95% CI: 1.11-2.17), and urothelial cancers (OR: 1.36; 95% CI: 1.01-1.73). Moreover, a higher level of DII is in association with a higher risk for mortality caused by all types of cancer by 16% (OR: 1.16; 95% CI: 1.01-1.32). In addition, meta-regression analysis reveals that the design of study can have a significant effect on the association of DII and incidence of all cancer types (slope: 0.54; $P = 0.05$). The stratified meta-analysis shows that the association of DII and incidence of all cancer types in case-control studies (OR: 1.53; 95% CI: 1.36-1.71) were more prominent than cohort studies (OR: 1.18; 95% CI: 1.07-1.30). **Conclusions:** This study shows that a higher level of DII is associated with a higher risk of incidence and mortality of all cancer types. The findings of the present study suggest that modifying inflammatory properties of dietary patterns can reduce the risk of incidence and mortality of all cancer types.

Keyword: *Cancer, diet, dietary inflammatory index, inflammation*

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Background

Inflammation is now widely known as an adaptive pathophysiological response underlying various chronic diseases including type 2 diabetes mellitus, cardiovascular disease, obesity, metabolic diseases, and specific types of cancer.[1-3] Several factors are associated with inflammation such as sex, age, and lifestyle. Lifestyle such as diet, physical activity, and smoking as malleable factors can reduce inflammation and thereby contributing to health.

Diet plays a contributing role in the regulation of inflammatory process. Various biomarkers have used to evaluate the association of nutrition and low-grade inflammatory status.[4] Consequently, it may be beneficial to identify dietary patterns related to their inflammatory properties.[5] Dietary inflammatory index (DII) is a new approach used to evaluate the inflammatory potential of a diet as either anti-inflammatory or pro-inflammatory.[6] In fact, some of the dietary patterns such as western pattern diet rich in red meat and refined grains is associated with a higher level of CRP, TNF- α, IL-1β, IL-2, and IL-6, which is often referred to as pro-inflammatory biomarkers. In contrast, there is an inverse association between Mediterranean diet including high amounts of fruits, whole grains, extra-virgin olive oil, and pro-inflammatory status.[7,8]

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Nowadays, the inflammatory properties of diet and its role in preventing chronic diseases have attracted much attention from health sciences researchers. Although in recent years several studies have shown the association of DII and risk of different cancer types, the findings of these studies are heterogeneous according to the type of study and cancer. However, according to our knowledge, pooled estimate of association of DII and all cancers is unclear and have not been investigated yet by systematic review. The aim of this meta-analysis was to investigate the association of DII with risk of incidence and mortality of any cancer types.

Methods

To evaluate the maximum level of sensitivity, we simultaneously searched main international electronic data sources; PubMed and NLM Gateway (for MEDLINE), Institute of Scientific Information (ISI), and SCOPUS for studies until January, 2017. Further, a hand-search of all references included in the identified articles. We did not limit our research by the publication date and language.

Our strategy for searching relevant studies was using the following key words "Index-based dietary patterns," "dietary inflammatory Index or DII," and all related domains to neoplasm," "cancer," "Malignancy," and "tumor".

Any observational epidemiologic study, either cross-sectional, case-control, or cohort, which had used DII, and the estimation of a adjusted effect size measure [odds ratio (OR), relative risk (RR), and hazard ratio (HR)] and 95% confidence interval (CI) comparing level and score of the DII with respect to the risk of incidence, mortality, and length of hospitalization of all cancer types were eligible to include in this systematic review. We excluded all papers with duplicate entries. In case of multiple publications on the same population, only the largest study or the main source of data was included.

The quality of studies was assessed using the Newcastle-Ottawa scale designing for cohort and case-control studies. According to this scale, 9 points can be allocated to each study including four scores for selection, two scores for comparability, and three scores for assessment of outcomes. The process of quality assessment and data extraction was carried out independently by two research experts. Quality assessment agreement on quality assessment between raters was established using Cohen's kappa statistic. The Kappa statistic for agreement on quality assessment was 0.92, which shows perfect agreement. The discrepancy between the raters was resolved by an auditor. Data were extracted according to a checklist. The items on the checklist included (a) the number of citation; (b) demographic characteristics of population such as age, target population, and type of cancers; (c) methodological information of study such as study design, food assessment questionnaire, duration of follow-up, sample size, type of effect size measure (OR, RR, and HR), and adjusted covariates.

Statistical analysis

We examined the association of DII and cancers in terms of morbidity (incidence), mortality, and length of hospitalization. For meta-analysis, we classified cancers into four main categories: (a) digestive tract cancers; (b) hormone-dependent cancers; (c) respiratory tract cancers; and (d) urothelial cancers. However, for those studies that reported several adjusted models, we included only the multivariate model. Although in this systematic review we included all studies with reported DII as continuous (score) or categorical variable (tertile/quartile/quintile), we performed meta-analysis only for DII as categorical variable. In meta-analysis, risk of incidence and mortality of cancer in the highest level of DII (last tertile/quartile/quintile) was compared with lowest level of DII (last tertile/quartile/quintile). Although a number of studies have reported cancer subsites, meta-analysis have not performed according to subsites of cancer.[9-14] The meta-analysis on the association between DII and risk of cancer mortality has been conducted only for all cancer mortality. Because there was only one study on the association between length of hospitalization and DII, we did perform mate-analysis for the association of DII and length of hospitalization of cancer.

The results reported as adjusted effect size measure and 95% CI. The Chi-square based Q test and I square statistics used to assess the heterogeneity between studies. The results of Q test were statistically significant at $P < 0.1$. Because of severe heterogeneity among studies on the reported values, pooled estimate was estimated using random-effect meta-analysis model (using the Dersimonian and Laird method). The forest plot also was used to present the results of meta-analysis schematically. A random-effects meta-regression was performed using unrestricted

maximum likelihood method to evaluate the association of estimated effect size measure and potential confounders such as design of study, type of cancer, food assessment questionnaire, and publication year. Potential publication bias was assessed using Egger's weighted regression tests, and the results of Egger's test were statistically significant at $P < 0.1$. The funnel plot also was used to present the results of publication bias schematically. "Trim and fill" method was used to adjust the analysis for the effects of publication bias. All statistical analysis was performed using STATA 11 software.

Ethical considerations

The protocol of study was approved by the ethical committee of Alborz University of Medical Science. All reviewed studies were properly cited. For more information about a certain study, we contacted the corresponding authors.

Results

The literature search strategy yielded a total of 575 publications. Further, 148 duplicated articles were excluded. After screening titles and abstracts, 345 irrelevant publications were excluded. Then, 82 remained articles and 6 retrieved articles through reference checking were carefully assessed and reviewed for eligibility; of which, 50 studies were excluded according to inclusion criteria. Finally, 38 studies met the inclusion criteria [Figure 1]. The main results of the selected articles were discussed

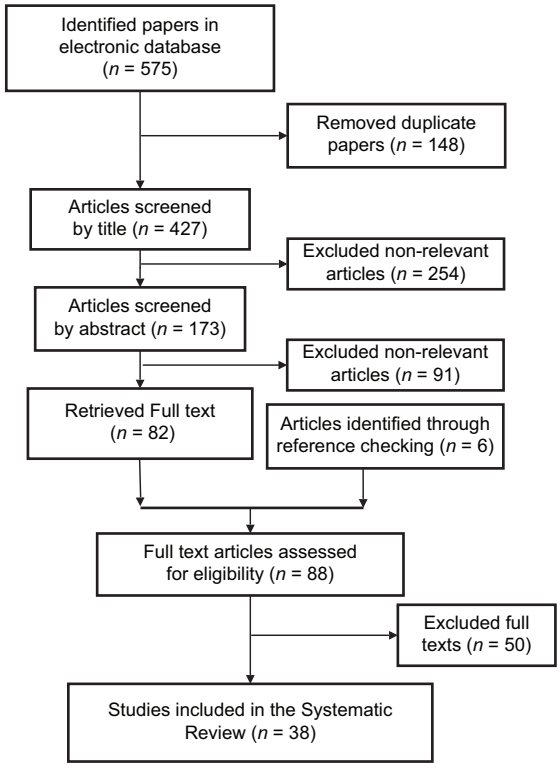

Figure 1: Papers search and review flowchart for selection of primary studies

in terms of incidence (n = 29), mortality (n = 7), both of them (n = 1), and length of hospitalization (n = 1) in patients with different types of cancers.

We found 30 articles (i.e. 20 case-controls and 10 cohorts) on the association of DII and incidence of different cancer types [Table 1]. Twenty-eight articles used food frequency questionnaire (FFQ), and the rest used 24 hour dietary recall (24HR) and dietary history questionnaire as dietary assessment instruments. The highest and lowest effect size measures (95% CI) were observed for esophageal squamous cell carcinoma (OR: 8.24; 95% CI: 2.03-33.47) and breast cancer (HR: 0.85; 95% CI: 0.52-1.41), respectively.

Table 2 summarizes 8 cohort studies on the association of DII and mortality of different cancer types. Dietary intake was measured using FFQ and 24HR in the five and three articles, respectively.

We found only one cohort study [Table 3] on the association between DII and length of hospitalization. There was no significant association exists between DII and length of hospitalization in surgical patients treated for colorectal cancer.

Table 4 presents the results of meta-analysis for the association of DII and incidence and mortality of different cancer types. There is a significant association between DII and incidence for all cancer types (OR: 1.32; 95% CI: 1.22-1.42; $P < 0.001$). A stratified meta-analysis by types of cancer shows that the highest and lowest effect size measures were observed for respiratory tract cancers and hormone-dependent cancers, respectively (OR: 1.64; 95% CI: 1.10-2.17 vs. OR: 1.14; 95% CI: 1.04-1.24). A stratified meta-analysis according to study design shows that the association of DII and incidence of all cancer types in case-control studies (OR: 1.53; 95% CI: 1.36-1.71) were more prominent than cohort studies (OR: 1.18; 95% CI: 1.07-1.30). Figures 2 and 3 report the forest plot of association between DII and cancer incidence according to the design of study and type of cancers, respectively. Moreover, there is a significant association between DII and mortality for all cancer types (HR: 1.16; 95% CI: 1.01-1.32) [Figure 4].

Meta-regression

A meta-regression analysis suggests that design of study can have a significant effect on the association between DII and cancer incidence (slope: 0.54; $P = 0.05$), whereas meta-regression does not show any significant associations between DII and type of food assessment questionnaire (slope:-0.33;p = 0.21), type of cancer (slope:-0.22; $P = 0.22$), and publication year (slope: 0.24;p = 0.31). The result of meta-regression analysis for the association of DII and cancer mortality shows no significant association between DII and type of food assessment questionnaire (slope: 0.43; $P = 0.47$), type of cancer (slope:-0.54; $P = 0.81$), and publication year (slope: 0.21; $P = 0.59$).

Table 1: Association between DII and risk of cancer incidence

Study number	First author (year)	Design	Follow--up (years)	Food assessment questionnaire	Type/site of cancer	Total sample size (incident cases)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
1	Samuel O. Antwi (2016)[30]	Case-control	NA	144 -item FFQ	Pancreatic cancer	2573 (817)	Quintile 5 (>-0.03, 4.47) vs. Quintile 1 (-5.33,-3.07)	OR	2.54 (1.87-3.46)	Age, sex, race, diabetes, BMI, pack-years of smoking, education
2	Young Ae Cho (2016)[9]	Case-control	NA	106-item semi-quantitative FFQ	Colorectal cancer Colon cancer Proximal colon cancer Distal colon cancer Rectal cancer	2769 (923) 2306 (460) 2011 (165) 2141 (295) 2290 (444)	Tertile 3 (≥2.30) vs. Tertile 1 (<0.30)	OR	2.16 (1.71-2.73) 2.05 (1.53-2.74) 1.68 (1.08-2.61) 2.28 (1.61-3.21) 2.23 (1.66-3.00)	age, sex, BMI, education, family history of colorectal cancer, physical activity, and total calorie intake
3-1	Pierre-Antoine Dugue (2016)[31]	cohort	21.3	121-item FFQ	Urothelial cell carcinoma	41514 (379)	Quintile 5 vs. Quintile 1*	HR	1.24 (0.90-1.70)	sex, country of birth, smoking, alcohol consumption, body mass index physical activity, education, and socioeconomic status
3-2	Pierre-Antoine Dugue (2016)[31]	cohort	21.3	121-item FFQ	Urothelial cell carcinoma	41514 (379)	Continuous DII (per one unit increment)	HR	1.07 (0.97-1.19)	sex, country of birth, smoking, alcohol consumption, body mass index physical activity, education, and socio-economic status
4	Isabell Ge (2015)[32]	case-control	NA	176-items FFQ	Breast cancer	8300 (2887)	Quintile 5 (1.922, 5.504) vs. Quintile 1 (-4.604, -0.213)	OR	1.01 (0.86-1.17)	age, study region, lifestyle confounders (total physical activity after 50 years, energy intake), breast cancer risk factors (age of menarche, number of pregnancies, breastfeeding history, induction of menopause, first-degree family history of breast cancer, history of benign breast disease, number of mammograms, hormone use)

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Study number	First author (year)	Design	Follow--up (years)	Food assessment questionnaire	Type/site of cancer	Total sample size (incident cases)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
5	Laurie Graffouille`re (2016)[a][33]	cohort	12.6	24 HR	Breast cancer	3771 (158)	Quartile 4 vs. Quartile 1*	HR	0.85 (0.52-1.41)	Age, sex, intervention group of the initial SU.VI. MAX trial, number of 24-h dietary records, BMI, height, physical activity, smoking status, educational level, energy intake, and family history in addition to menopausal status
					Prostate cancer	2771 (123)			2.08 (1.06-4.09)	
					vnon-prostate cancer (other cancers)	6542 (278)			1.34 (0.92-1.95)	
					All cancers	6542 (559)			1.23 (0.94-1.62)	
6	A. M. Hodge (2016)[26]	cohort	18	121-item FFQ	Lung cancer	35,303 (403)	Quartile 4 (0.39,4.86) vs. Quartile 1 (-4.91,-2.15)	HR	1.31 (0.91-1.89)	pack-years, years since quit smoking, smoking status, country of birth, education, BMI, alcohol intake, physical activity, sex, SEIFA quintile, energy (includes an interaction between smoking status and country of birth)
7	Yunxia Lu (2016)[34]	Case-control	NA	63-item FFQ	Esophageal squamous cell carcinoma	946 (158)	Quartile 4 (≥1.46) vs. Quartile 1 (<−1.04)	OR	4.35 (2.24-8.43)	age, sex, energy, education, tobacco smoking, alcohol intake, and physical activity in addition to reflux, and Helicobacter pylori infection (for oesophageal adenocarcinoma and gastroesophageal junctional adenocarcinoma)
					Esophageal adenocarcinoma	987 (181)			3.59 (1.87-6.89)	
					Gastroesophageal junctional adenocarcinoma	1061 (255)			2.04 (1.24-3.36)	
					Esophageal or gastroesophageal junction adenocarcinoma	1242 (436)			2.42 (1.57-3.73)	
8	Patrick Maisonneuve (2016)[35]	Cohort	8.5	45-item FFQ	Lung cancer	4336 (200)	Quartile 4 vs. Quartile 1*	HR	1.54 (0.93-2.55)	baseline risk probability (age, sex, smoking duration, smoking intensity, years of smoking cessation, and asbestos exposure) and total energy

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Study number	First author (year)	Design	Follow--up (years)	Food assessment questionnaire	Type/site of cancer	Total sample size (incident cases)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
9-1	Lauren C. Peres (2017)[36]	case-control	NA	110-item FFQ	Epithelial ovarian cancer	1155 (493)	Quartile 4 (-0.32, 3.19) vs. Quartile 1 (-5.57, -3.64)	OR	1.72 (1.18-2.51)	study design variables, age, and study site, family history of breast or ovarian cancer in a first degree relative, parity, OC use, education, BMI, tubal ligation, menopausal status, smoking status, and endometriosis
9-2	Lauren C.Peres (2017)[36]	case-control	NA	110-item FFQ	Epithelial ovarian cancer	1155 (493)	Continuous DII (per one unit increment)	OR	1.10 (1.03-1.17)	study design variables, age and study site, family history of breast or ovarian cancer in a first degree relative, parity, OC use, education, BMI, tubal ligation, menopausal status, smoking status, and endometriosis
10-1	Nitin Shivappa (2016)[a][37]	Cohort	25	121-item FFQ	Breast cancer	34700 (2934)	Tertile 3 (> -0.05) vs.Tertile 1 (<-2.08)	HR	1.11 (1.00-1.22)	Age, energy and BMI, smoking status, pack-years of smoking, education, HRT use, oral contraceptive use, number of live births, education, age at menarche, age at menopause and history of hysterectomy
10-2	Nitin Shivappa (2016)[a][37]	Cohort	25	121-item FFQ	Breast cancer	34700 (2934)	Continuous DII (per one unit increment)	HR	1.01 (0.99-1.04)	Age, energy and BMI, smoking status, pack-years of smoking, education, HRT use, oral contraceptive use, number of live births, education, age at menarche, age at menopause and history of hysterectomy

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Table 1: Contd...

Study number	First author (year)	Design	Follow--up (years)	Food assessment questionnaire	Type/site of cancer	Total sample size (incident cases)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
11	Nitin Shivappa (2015)[c][38]	case-control	NA	78-item FFQ	Prostate cancer	2754 (1294)	Quartile 4 (>0.49) vs. Quartile 1 (<-1.98)	OR	1.33 (1.01-1.76)	Age, study center, BMI, years of education, social class, smoking status, family history of prostate cancer, and total energy intake
12	Nitin Shivappa (2015)[d][39]	case-control	NA	78-item FFQ	Pancreatic cancer	978 (326)	Quintile 5 (≥ 1.27) vs.Quintile 1 (< - 1.28)	OR	2·48 (1.50-4.10)	Age, sex, study center, year of interview, education, BMI, smoking status, alcohol drinking, and history of diabetes
13-1	Nitin Shivappa (2016)[f][40]	case-control	NA	78-item FFQ	Gastric cancer	777 (230)	Quartile 4 (>1.49) vs. Quartile 1 (≤1.47)	OR	2.35 (1.32-4.20)	study center, age, education, year of interview, BMI, smoking and total energy intake
13-2	Nitin Shivappa (2016)[f][40]	case-control	NA	78-item FFQ	Gastric cancer	777 (230)	Continuous DII (per one unit increment)	OR	1.19 (1.06-1.34)	study center, age, education, year of interview, BMI, smoking, and total energy intake
14-1	Nitin Shivappa (2015)[g][41]	case-control	NA	125-item FFQ	Esophageal squamous cell carcinoma	143 (47)	High (>1.20) vs. Low (≤120)	OR	8.24 (2.03-33.47)	age, energy, sex, BMI, years of education, physical activity, smoking, and gastro-oesophageal reflux
14-2	Nitin Shivappa (2015)[g][41]	case-control	NA	125-item FFQ	Esophageal squamous cell carcinoma	143 (47)	Continuous DII (per one unit increment)	OR	3.58 (1.76-7.26)	age, energy, sex, BMI, years of education, physical activity, smoking, and gastro-oesophageal reflux
15-1	Nitin Shivappa (2016)[h][42]	case-control	NA	78-item FFQ	Breast cancer	5157 (2569)	Quintile 5 (1.28, 5.14) vs.Quintile 1 (-6.18,-2.13)	OR	1.75 (1.39-2.21)	age, study center, and energy intake, education, body mass index, parity, menopausal status, and family history of hormone-related cancers

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Study number	First author (year)	Design	Follow--up (years)	Food assessment questionnaire	Type/site of cancer	Total sample size (incident cases)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
15-2	Nitin Shivappa (2016)[h][42]	case-control	NA	78-item FFQ	Breast cancer	5157 (2569)	Continuous DII (per one unit increment)	OR	1.09 (1.05-1.14)	age, study center, and energy intake, education, body mass index, parity, menopausal status, and family history of hormone-related cancers
16-1	Nitin Shivappa (2016)[i][43]	case-control	NA	80-item FFQ	Bladder Cancer	1355 (690)	Quartile 4 (0.42, 4.58) vs.Quartile 1 (-5.94,-2.41)	OR	1.97 (1.28-3.03)	age, sex, year of interview, study center, total energy intake, education, and tobacco smoking
16-2	Nitin Shivappa (2016)[i][43]	case-control	NA	80-item FFQ	Bladder Cancer	1355 (690)	Continuous DII (per one unit increment)	OR	1.11 (1.03-1.20)	age, sex, year of interview, study center, total energy intake, education, and tobacco smoking
17-1	Nitin Shivappa (2016)[j][27]	case-control	NA	78-item FFQ	ovarian cancer	3442 (1031)	Quartile 4 (>1.35) vs. Quartile 1 (≤1.63)	OR	1.47 (1.07-2.01)	age, energy intake, year of interview, study center, education, body mass index, parity, oral contraceptive use, menopausal status, and family history of ovarian and/or breast cancer in first-degree relatives
17-2	Nitin Shivappa (2016)[j][27]	case-control	NA	78-item FFQ	Ovarian cancer	3442 (1031)	Continuous DII (per one unit increment)	OR	1.08 (1.02-1.14)	age, energy intake, year of interview, study center, education, body mass index, parity, oral contraceptive use, menopausal status, and family history of ovarian and/or breast cancer in first-degree relatives
18-1	Nitin Shivappa (2016)[k][44]	case-control	NA	78-item FFQ	Laryngeal cancer	1548 (460)	Quartile 4 (0.27, 5.00) vs.Quartile 1 (-5.48,-2.19)	OR	3.30 (2.06-5.28)	age, sex, center, education, body mass index, tobacco smoking, alcohol consumption, and non-alcohol energy intake

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Study number	First author (year)	Design	Follow- -up (years)	Food assessment questionnaire	Type/site of cancer	Total sample size (incident cases)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
18-2	Nitin Shivappa (2016)[k][44]	case-control	NA	78-item FFQ	Laryngeal cancer	1548 (460)	Continuous DII (per one unit increment)	OR	1.27 (1.15, 1.40)	age, sex, center, education, body mass index, tobacco smoking, alcohol consumption, and non-alcohol energy intake
19-1	Nitin Shivappa (2016)[l][45]	case-control	NA	78-item FFQ	Nasopharyngeal cancer	792 (198)	Tertile 3 (men: >0.59; women: >−0.19) vs. Tertile1 (men: ≤−0.64; women: ≤−1.06)	OR	1.64 (1.06-2.55)	place of living, sex, age, year of interview, education, smoking, alcohol drinking, and energy intake according to the residual method
19-2	Nitin Shivappa (2016)[l][45]	case-control	NA	78-item FFQ	Nasopharyngeal cancer	792 (198)	Continuous DII (per one unit increment)	OR	1.19 (1.05, 1.36)	place of living, sex, age, year of interview, education, smoking, alcohol drinking, and energy intake according to the residual method
20-1	Nitin Shivappa (2016)[m][46]	case-control	NA	78-item FFQ	Endometrial cancer	1362 (454)	Quartile 4 (>1·04) vs. Quartile 1 (<−1·07)	OR	1·46 (1·02-2·11)	age, energy, year of interview, education, BMI, age at menarche, menopausal status and age at menopause, parity, history of diabetes, family history of cancers, oral contraceptive use and hormone replacement therapy use
20-2	Nitin Shivappa (2016)[m][46]	case-control	NA	78-item FFQ	Endometrial cancer	1362 (454)	Continuous DII (per one unit increment)	OR	1.07 (0.98-1.17)	age, energy, year of interview, education, BMI, age at menarche, menopausal status and age at menopause, parity, history of diabetes, family history of cancers, oral contraceptive use and hormone replacement therapy use

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Table 1: Contd...

Study number	First author (year)	Design	Follow--up (years)	Food assessment questionnaire	Type/site of cancer	Total sample size (incident cases)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
21-1	Nitin Shivappa (2015)[n][47]	case-control	NA	21-item FFQ	Prostate cancer	479 (229)	Quartile 4 vs. Quartile 1*	OR	2.39 (1.14-5.04)	age, BMI, smoking status, education, physical activity, energy intake, family history of prostate cancer
21-2	Nitin Shivappa (2015)[n][47]	case-control	NA	21-item FFQ	Prostate cancer	479 (229)	Continuous DII (per one unit increment)	OR	1.27 (0.98-1.50)	age, BMI, smoking status, education, physical activity, energy intake, and family history of prostate cancer
22-1	Nitin Shivappa (2014)[o][10]	Cohort	19.6±7.0	121-item FFQ	Colorectal cancer	34703 (1636)	Quintile 5 (>1.10) vs. Quintile 1 (<-2.75)	HR	1.20 (1.01-1.43)	age, BMI, smoking status, pack-years of smoking, HRT use, education, diabetes, and total energy intake
					Colon cancer	34703 (1329)			1.19 (0.98-1.45)	
					Rectal cancer	34703 (325)			1.21 (0.81-1.79)	
22-2	Nitin Shivappa (2014)[o][10]	Cohort	19.6±7.0	121-item FFQ	Colorectal cancer	34703 (1636)	Continuous DII (per one unit increment)	HR	1.07 (1.01-1.13)	age, BMI, smoking status, pack-years of smoking, HRT use, education, diabetes, and total energy intake
					Colon cancer	34703 (1329)			1.05 (0.99-1.12)	
					Rectal cancer	34703 (325)			1.11 (0.98-1.25)	
23-1	Nitin Shivappa (2015)[p][48]	Cohort	20	80-item FFQ	Breast cancer	45257 (1895)	Quartile 4 (>3.77) vs. Quartile 1 (<1.87)	HR	1.18 (1.00-1.39)	age, energy, age at first birth and number of children, age at menarche, BMI, height, multivitamin use, education, smoking status, oral contraceptive use, and family history of breast cancer in the model
23-2	Nitin Shivappa (2015)[p][48]	Cohort	20	80-item FFQ	Breast cancer	45257 (1895)	Continuous DII (per one unit increment)	HR	1.04 (1.01-1.09)	age, energy, age at first birth and number of children, age at menarche, BMI, height, multivitamin use, education, smoking status, oral contraceptive use, and family history of breast cancer in the model

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Table 1: Contd...

Study number	First author (year)	Design	Follow--up (years)	Food assessment questionnaire	Type/site of cancer	Total sample size (incident cases)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
24-1	Nitin Shivappa (2015)[r [11]]	case-control	NA	78-item FFQ	Colorectal cancer	6107 (1953)	Quintile 5 (>1.22) vs. Quintile 1 (≤ -1·05)	OR	1.55 (1.29-1.85)	age, sex, study center, education, BMI, alcohol drinking, physical activity, and history of colorectal cancer and energy intake (using the residual method)
					Colon cancer	5379 (1225)			1·39 (1.13-1.71)	
					Rectal cancer	4882 (728)			1·47 (1.14-1.90)	
24-2	Nitin Shivappa (2015)[r [11]]	Case-control	NA	78-item FFQ	Colorectal cancer	6107 (1953)	Continuous DII (per one unit increment)	OR	1·13 (1·09-1·18)	age, sex, study center, education, BMI, alcohol drinking, physical activity, and history of colorectal cancer and energy intake (using the residual method)
					Colon cancer	5379 (1225)			1·09 (1·04, 1·14)	
					Rectal cancer	4882 (728)			1·12 (1·06, 1·19)	
25-1	Nitin Shivappa (2015)[s [49]]	Case-control	NA	78-item FFQ	Esophageal squamous cell cancer	1047 (304)	Quintile 5 (>1.28) vs. Quintile 1 (<-1.20)	OR	2.47 (1.40-4.36)	age, sex, year of interview, and area of residence and adjusted for education, alcohol drinking, tobacco smoking, BMI, physical activity, aspirin use, and energy (using the residual method)
25-2	Nitin Shivappa (2015)[s [49]]	Case-control	NA	78-item FFQ	Esophageal squamous cell cancer	1047 (304)	Continuous DII (per one unit increment)	OR	1.23 (1.10-1.38)	age, sex, year of interview, and area of residence and adjusted for education, alcohol drinking, tobacco smoking, BMI, physical activity, aspirin use, and energy (using the residual method)

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Table 1: Contd...

Study number	First author (year)	Design	Follow--up (years)	Food assessment questionnaire	Type/site of cancer	Total sample size (incident cases)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
26	Fred K Tabung (2016)[a] [50]	Cohort	16.02	122-item FFQ	Breast cancer	122788 (7495)	Quintile 5 (1.898,5.519) vs. Quintile 1 (-7.055,< -3.142)	HR	0.99 (0.91-1.07)	age, energy intake, race/ethnicity, income, education, smoking status, mammography within 2 years of baseline, age at menarche, number of live births, oophorectomy status, hormone therapy use, nonsteroidal anti-inflammatory drug (NSAID) use, dietary modification trial arm, hormone therapy trial arm, body mass index, and physical activity
27	Fred K Tabung (2015)[b] [12]	Cohort	11.3	122-item FFQ	Colorectal cancer	152,536 (1920)	Quintile 5 (1.953, 5.636) vs. Quintile 1 (-7.055, < -3.136)	HR	1.22 (1.05-1.43)	age, total energy intake, body mass index, race/ethnicity, physical activity, educational level, smoking status, family history of colorectal cancer, hypertension, diabetes, arthritis, history of colonoscopy, history of occult blood tests, NSAID use, category and duration of estrogen use, category and duration of estrogen and progesterone use, dietary modification trial arm, hormone therapy trial arm and calcium and vitamin DMT trial arm
					Colon cancer	152,536 (1559)			1.23 (1.03-1.46)	
					Proximal colon cancer	152,536 (1034)			1.35 (1.09-1.67)	
					Distal colon cancer	152,536 (428)			0.84 (0.61-1.18)	
					Rectal cancer	152,536 (361)			1.20 (0.84-1.72)	

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Table 1: Contd...

Study number	First author (year)	Design	Follow--up (years)	Food assessment questionnaire	Type/site of cancer	Total sample size (incident cases)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
28-1	Ruth A. Vázquez-Salas (2016)[51]	Case-control	NA	127-item semi-quantitative FFQ	Prostate cancer	1188 (394)	Tertile 1 (ref) (<−0·12) vs. Tertile 3 (≥1·28) Continuous DII (per…)	OR	1.18 (0.85-1.63)	age, educational level, history of PC in first-degree relatives, BMI 2 years before the interview, physical activity throughout life, smoking status 5 years before the interview,history of chronic diseases
28-2	Ruth A. Vázquez-Salas (2016) [51]	Case-control	NA	127-itemsemi -quantitative FFQ	Prostate cancer	1188 (394)	Continuous DII (per one unit increment)	OR	1·02 (0·94, 1·11)	age, educational level, history of PC in first-degree relatives, BMI 2 years before the interview, physical activity throughout life, smoking status 5 years before the interview, history of chronic diseases
29-1	Michael D. Wirth (2015)[13]	Cohort	9.1±2.9	124-item FFQ	Colorectal cancer	489,442 (6225)	Quartile 4 (3.25, 6.97) vs. Quartile 1 (-7.33,-0.59)	HR	1.40 (1.28-1.53)	age, smoking status, BMI, self-reported diabetes, and energy intake - for 1:physical activity, marital status, education and age (STRATA statement) - for 2:age (STRATA statement) -For 3:race and age -For 4:marital status, education, perceived health, census-based income and age (STRATA statement) -for 5:self-reported polyps, education, age and census-based income
					Ascending/ Cecum	489,442 (2060)			1.27 (1.09-1.49)	
					Transverse/ Hepatic and Splenic Flexure	489,442 (802)			1.58 (1.23-2.03)	
					Descending/ Sigmoid	489,442 (1614)			1.61 (1.35-1.91)	
					Rectum/Recto sigmoid	489,442 (1680)			1.45 (1.22-1.73)	

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Table 1: Contd...

Study number	First author (year)	Design	Follow--up (years)	Food assessment questionnaire	Type/site of cancer	Total sample size (incident cases)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
29-2	Michael D. Wirth (2015) [13]	Cohort	9.1±2.9	124-item FFQ	Colorectal cancer	489,442 (6225)	Continuous DII (per one unit increment)	HR	1.06 (1.05-1.08)	age, smoking status, BMI, self-reported diabetes, and energy intake - for 1:physical activity, marital status, education and age (STRATA statement) - for 2:age (STRATA statement) -For 3:race and age -For 4:marital status, education, perceived health, census-based income and age (STRATA statement) -for 5:self-reported polyps, education, age and census-based income
					Ascending/ Cecum	489,442 (2060)			1.05 (1.02-1.07)	
					Transverse/ Hepatic and Splenic Flexure	489,442 (802)			1.06 (1.02-1.10)	
					Descending/ Sigmoid	489,442 (1614)			1.08 (1.05-1.11)	
					Rectum/Recto sigmoid	489,442 (1680)			1.08 (1.05-1.10)	
30-1	Raul Zamora-Ros (2015)[14]	Case-control	NA	dietary history questionnaire	Colorectal cancer	825 (424)	Quartile 4 (>3.05) vs. Quartile 1 (<-0.73)	OR	1.65 (1.05-2.60)	sex, age, total energy intake, BMI, first-degree family history of colorectal cancer, physical activity, tobacco consumption, and medication use (aspirin and non-steroidal anti-inflammatory drug)
					Colon cancer	666 (265)			2.24 (1.33-3.77)	
					Rectal cancer	560 (159)			1.12 (0.61-2.06)	
30-2	Raul Zamora-Ros (2015)[14]	Case-control	NA	dietary history questionnaire	Colorectal cancer	825 (424)	Continuous DII (per one unit increment)	OR	1.08 (1.01-1.15)	sex, age, total energy intake, BMI, first-degree family history of colorectal cancer, physical activity, tobacco consumption, and medication use (aspirin and non-steroidal anti-inflammatory drug
					Colon cancer	666 (265)			1.12 (1.04-1.21)	
					Rectal cancer	560 (159)			1.03 (0.95-1.12)	

Abbreviation: FFQ: food frequency questionnaire, 24HR: 24 hour recall, HR: hazard ratio, OR: odds ratio; DII: dietary inflammatory index; NA: not applicable

Table 2: Association of DII and risk of cancer mortality

Study number	First author (year)	design	Follow up (years)	Food assessment questionnaire	Study subjects	Type of cancer mortality	Total sample size (death number)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
1-1	Fang Emily Deng (2016)[52]	cohort	135 and 168 person -months	24 HR	Normal	Allcancers	9631 (385)	Tertile 1 (ref) (<−0.20)	HR	1.23 (0.84-1.79)	age, sex, race, HgbA1C, current
						Lung cancer	9631 (99)	vs. Tertile 3 (>2.0)		1.4 (0.79-2.47)	smoking,physical activity, BMI, SBP
						Digestive-tract cancer	9631 (99)			1.38 (0.69-2.76)	
1-2	Fang Emily Deng (2016)[52]	cohort	135 and 168 person -months	24 HR	Pre -diabetic	All cancers	2681 (208)	Tertile 1 (ref) (<−0.20)	HR	2.02 (1.27-3.21)	age, sex, race, HgbA1C, current
						Lung cancer	2681 (66)	vs. Tertile 3 (>2.0)		2.01 (0.93-4.34)	smoking, physical activity, BMI, SBP
						Digestive-tract cancer	2681 (50)			2.89 (1.08-7.71)	
1-3	Fang Emily Deng (2016)[52]	cohort	135 and 168 person -months	24 HR	Diabetic	All cancers	968 (83)	Tertile 1 (ref) (<−0.20)	HR	1.00 (0.49-2.04)	age, sex, race, HgbA1C, current
						Lung cancer	968 (27)	vs. Tertile 3 (>2.0)		0.55 (0.09-3.36)	smoking, physical activity, BMI, SBP
						Digestive-tract cancer	968 (27)			1.30 (0.40-4.28)	
2-1	Aleksander Galas (2014)a[53]	cohort	3,180.31 person -years	148 item semi -quantitative FFQ	Patients without distant metastases	Colorectal cancer	511 (150)	High (>- 2.27) vs. low (≤ -2.27)	HR	0.76 (0.55-1.08)	Age, smoking, marital status, overweight or obesity, calendar year when surgery was performed, surgery type, cancer site, chemotherapy after surgery, radiotherapy after surgery
					Patients with distant metastases		178 (159)			1.06 (0.76-1.48)	
2-2	Aleksander Galas (2014)a[53]	cohort	3,180.31 person -years	148 itemsemi -quantitative FFQ	Patients without distant metastases	Colorectal cancer	511 (150)	Continuous DII (per one unit increment)	HR	0.98 (0.92-1.05)	Age, smoking, marital status, overweight or obesity, calendar year when surgery was performed, surgery type, cancer site, chemotherapy after surgery, radiotherapy after surgery
					Patients with distant metastases		178 (159)			1.003 (0.93-1.08)	

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Table 2: Contd...

Study number	First author (year)	design	Follow up (years)	Food assessment questionnaire	Study subjects	Type of cancer mortality	Total sample size (death number)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
3-1	Laurie Graffouille`re (2016)[b][54]	cohort	12.4	24 HR	Healthy subjects	All cancers	7994 (123)	Tertile 3 vs. Tertile 1*	HR	1.83 (1.12-2.99)	Age, sex, intervention group of the initial SU.VI.MAX trial, number of 24-h dietary records, BMI, physical activity, smoking status, educational level, family history of cancer in first-degree relatives, family history of CVD in first-degree relatives, energy intake without alcohol, and alcohol intake
3-2	Laurie Graffouille`re (2016)[b][54]	cohort	12.4	24 HR	Healthy subjects	All cancers	7994 (123)	Continuous DII (per one unit increment)	HR	1.18 (1.04-1.34)	age, sex, intervention group of the initial SU.VI.MAX trial, number of 24-h dietary records, BMI, physical activity, smoking status, educational level, family history of cancer in first-degree relatives, family history of CVD in first-degree relatives, energy intake without alcohol, and alcohol intake
4-1	Nitin Shivappa (2016)[b][55]	Cohort	25	121-item FFQ	postmeno-pausal women	All cancers Digestive tract cancers	37525 (5044) 37525 (1240)	Quartile 4 (0.6469 to 4.6598) vs. Quartile 1(−5.7509 to−2.5041)	HR	1.08 (0.99-1.18) 1.19 (1.00-1.43)	age, BMI, smoking status, pack-years of smoking, HRT use, education, prevalent diabetes, prevalent hypertension, prevalent heart disease, prevalent cancer, total energy intake

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Table 2: Contd...

Study number	First author (year)	design	Follow up (years)	Food assessment questionnaire	Study subjects	Type of cancer mortality	Total sample size (death number)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
4-2	Nitin Shivappa (2016)[b][55]	Cohort	25	121-item FFQ	postmeno-pausal women	All cancers Digestive tract cancers	37525 (5044) 37525 (1240)	Continuous DII (per one unit increment)	HR	1.04 (1.01-1.07) 1.07 (1.01-1.14)	age, BMI, smoking status, pack-years of smoking, HRT use, education, prevalent diabetes, prevalent hypertension, prevalent heart disease,prevalent cancer, total energy intake
5-1	Nitin Shivappa (2016)[e][56]	Cohort	15	96-item FFQ	Healthy women	All cancers Digestive tract cancers	33747 (1996) 33747 (602)	Quintile 5 (> 5.10) vs. Quintile 1 (<−4.19)	HR	1.25 (0.96-1.64) 1.42 (0.82-2.49)	Age, energy, BMI, education, smoking status, physical activity, alcohol intake
5-2	Nitin Shivappa (2016)[e][56]	Cohort	15	96-item FFQ	Healthy women	All cancers Digestive cancer	33747 (1996) 33747 (602)	Continuous DII (per one unit increment)	HR	1.04 (0.99-1.11) 1.15 (1.02-1.29)	Age, energy, BMI, education, smoking status, physical activity, alcohol intake
6-1	Nitin Shivappa (2015)[q][57]	Cohort	13.5±4.0	24 HR	Healthy subjects	All cancers Digestive tract cancers	12366 (615) 12,366 (158)	Tertile 3 (2.03 to 4.83) vs. Tertile 1 (−5.60 to−0.22)	HR	1.46 (1.10-1.96) 2.10 (1.15-3.84)	age, sex, race, diabetes status, hypertension, physical activity, BMI, poverty index, and smoking
6-2	Nitin Shivappa (2015)[q][57]	Cohort	13.5±4.0	24 HR	Healthy subjects	All cancers Digestive tract cancers	12,366 (615) 12,366 (158)	Continuous DII (per one unit increment)	HR	1.04 (0.97-1.11) 1.08 (0.95-1.22)	age, sex, race, diabetes status, hypertension, physical activity, BMI, poverty index, and smoking

Contd...

Table 2: Contd...

Study number	First author (year)	design	Follow up (years)	Food assessment questionnaire	Study subjects	Type of cancer mortality	Total sample size (death number)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
7	Fred K Tabung (2016) [a][50]	Cohort	16.02	122-item FFQ	Postmeno-pausal women	Breast cancer	122788 (667)	Quintile 5 (1.874 to 5.519) vs. Quintile 1 (−7.055 to <−3.162)	HR	1.33 (1.01-1.76)	age, energy intake, race/ethnicity, income, education, smoking status, mammography within 2 years of baseline, age at menarche, number of live births, oophorectomy status, hormone therapy use, nonsteroidal anti-inflammatory drug (NSAID) use, dietary modification trial arm, hormone therapy trial arm, body mass index, and physical activity
8	Antonella Zucchetto (2016)[58]	Cohort	12.7	78-item FFQ	Patients with prostate cancer	Prostate cancer	726 (76)	Tertile 3 vs. Tertile 1*	HR	1.42 (0.73-2.76)	area of residence, calendar period of diagnosis, age at diagnosis, education, smoking habits, abdominal obesity, alcohol intake, energy intake

FFQ: Food frequency questionnaire, 24HR: 24 hour recall, HR: Hazard ratio, DII: Dietary inflammatory index

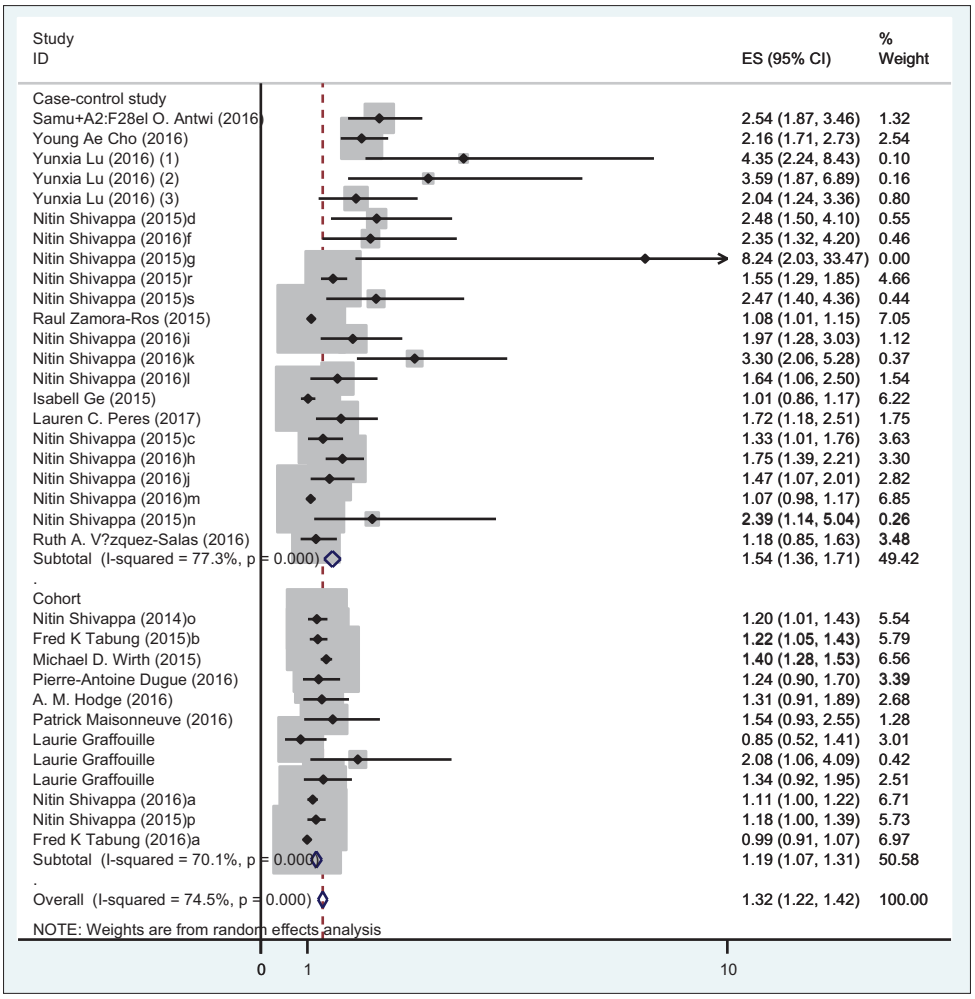

Figure 2: Odds ratio and 95% CI of individual studies and pooled data for the association between DII and incidence of cancer according to the type of study using random-effect model. OR: Odds of ratios

Table 3: Association between DII and length of hospitalization

Study number	First author (year)	design	Follow up (years)	Food assessment questionnaire	Study subjects	Type of cancer mortality	Total sample size (death number)	Groups	Type of effect size measure	Effect size measure (95% CI)	Covariates
1	Aleksander Galas (2014)[b][59]	Cohort	11 days	148 itemsemi-quantitative FFQ	Surgical patients treated for colorectal cancer	Colorectal cancer	689	Over the first tertile (> -3.41) vs. tertile 1 (≤ -3.41)	OR	0.76 (0.53-1.09)	Age, smoking, marital status, overweight or obesity, calendar year when surgery was performed, surgery type, cancer site, chemotherapy after surgery, radiotherapy after surgery
								Over the first quartile (> -3.91) vs. quartile 1 (≤ -3.91)		0.69 (0.46-1.03)	
								Over the first quintile (> -4.25) vs. quintile 1 (≤ -4.25)		0.69 (0.45-1.07)	

FFQ: Food frequency questionnaire, OR: Odds ratio

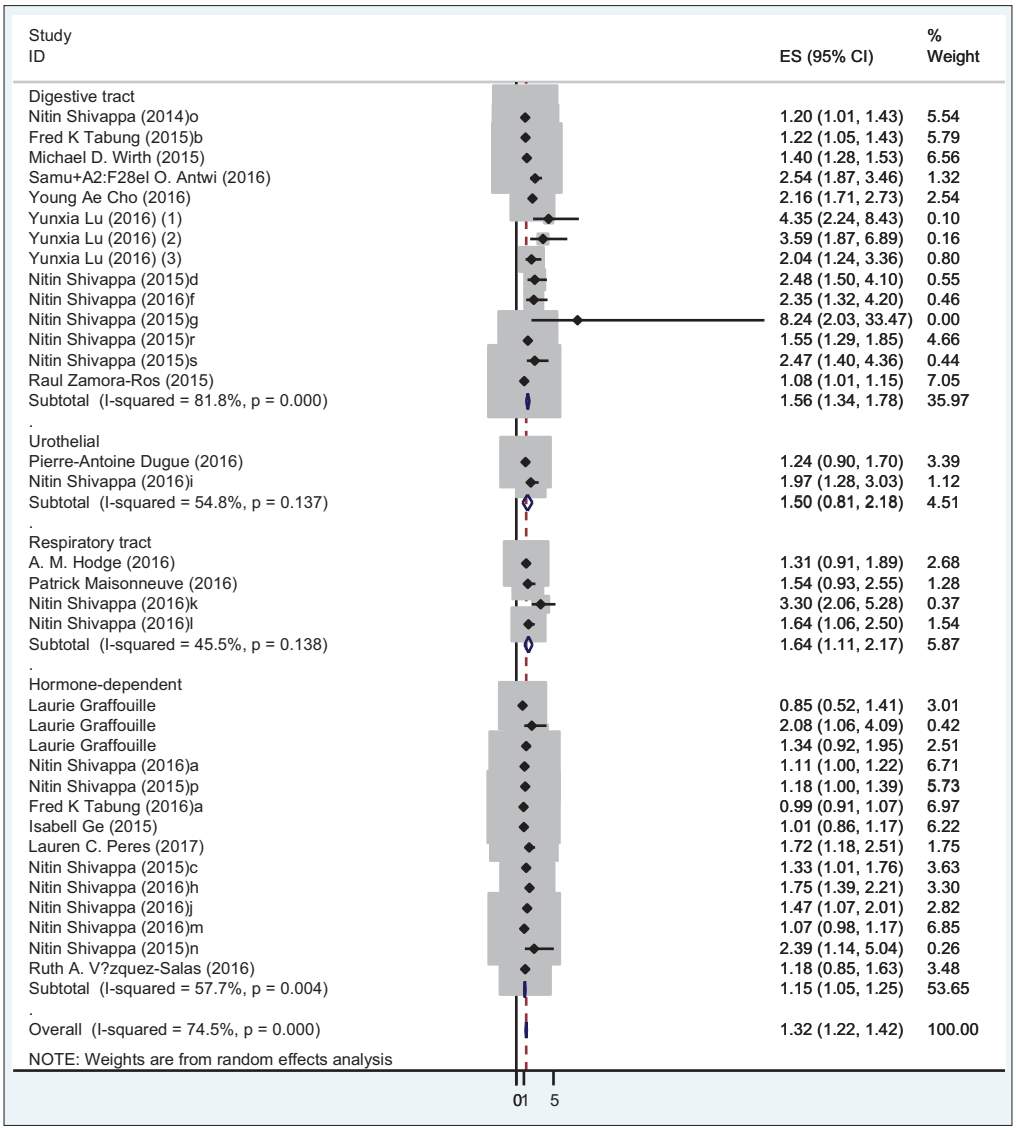

Figure 3: Odds ratio and 95% CI of individual studies and pooled data for the association between DII and incidence of cancer according to the type of cancer using random-effect model. OR: Odds of ratios

Table 4: Meta-analysis of association between DII and mortality/morbidity of cancer

Type of outcome (Mortality/morbidity)	subgroup	Type of cancer	Number of studies	Test of association			Test of heterogeneity			
				Effect size measure	95%CI	*P*	Model	I^2	Q test	*P*
Morbidity	Type of cancer	Digestive tract cancers	14	1.55	1.33-1.78	< 0.001	Random	81.8	71.27	< 0.001
		Hormone-dependent cancers	13	1.14	1.04-1.24	< 0.001	Random	59.6	29.72	0.003
		Respiratory tract cancers	4	1.64	1.11-2.17	< 0.001	Fixed	45.5	5.51	0.13
		Urothelial cancers	2	1.36	1.00-1.73	< 0.001	Fixed	54.8	2.21	0.13
	Type of study	Case-control	22	1.53	1.36-1.71	< 0.001	Random	77.3	92.51	< 0.001
		Cohort	12	1.18	1.07-1.30	< 0.001	Random	70.1	36.81	< 0.001
	Overall		34*	1.32	1.22-1.42	< 0.001	Random	74.5	129.39	< 0.001
Mortality	All cancers		11	1.16	1.01-1.32	< 0.001	Random	44.3	17.96	0.056

*The sum of number of studies for all cancers (34 studies) is more than the sum of digestive, hormone-dependent, respiratory and urothelial cancers because in one study, type of cancer was not reported

Publication bias

The results of Egger test for association of DII and all cancer incidence show that publication bias exists (coefficient: 2.87; $P < 0.001$) and funnel plot was asymmetric [Figure 5]. "Trim and fill" correction suggested some potentially missing study on the right side of funnel plot [Figure 5]. Imputation for this potentially missing study yielded an effect size of 1.23 (95% CI: 1.12-1.33). In addition, the results of Egger test for association between DII and all cancer mortality show that publication bias does not exist (coefficient: 1.06; $P = 0.15$) and funnel plot was symmetric [Figure 6].

Discussion

To the best of our knowledge, the present study is the first comprehensive systematic review and meta-analysis on the association of DII and cancer incidence and mortality. This meta-analysis shows a significant association between DII and risk of incidence and mortality of all cancer types. The results of the present study shows that a higher level of DII increases the risk of cancers incidence by 32% (95% CI: 1.22-1.42) including digestive tract cancers (OR: 1.55; 95% CI: 1.33-1.78), hormone-dependent cancers (OR = 1.14; 95% CI: 1.04-1.24), respiratory tract cancers (OR: 1.64; 95% CI: 1.11-2.17), and urothelial cancers (OR: 1.36; 95% CI: 1.00-1.73). Moreover, a higher level of DII in association with a higher risk of mortality caused by all type of cancer by 16% (95% CI: 1.01-1.32).

Our findings were consistent with previous studies showing that a higher DII was associated with mortality. Moreover, some studies have documented a direct association between DII and a higher risk for metabolic syndrome and cardiovascular diseases (CVD).[4] One of the study reported different mechanisms by which inflammatory markers used for DII calculation can predict most prevalent diseases including cancers, CVD, and diabetes.[6]

Results of present study show that the association of DII and incidence of all cancer types in case-control studies were more prominent than cohort studies, which was consistent with previous studies.[15,16] It has been suggested that dietary recall bias may justify the discrepant results between case-control and cohort studies on diet and the risk of cancers.

Dietary patterns analysis is one of the most appropriate approaches to understand the relationship between diet and risk for various diseases including diabetes, cancers, and CVD.[17] All of the healthy dietary patterns (e.g. Dietary Approaches to Stop Hypertension and Mediterranean diet) can play a key role in preventing major chronic diseases, especially cancers.[18-20] In contrast, there was an inverse relationship between DII and dietary quality indices (e.g. Healthy Eating Index).[21] This was in line with the number of studies showing an inverse correlation between C-reactive protein, one of the inflammatory biomarkers used to calculate the DII, and higher consumption of vegetables, fruits,[22] legumes,[23] and nuts.[24]

To define the inflammatory capacity of diet as a main determining factor for vast majority of chronic diseases, we developed DII from peer-reviewed

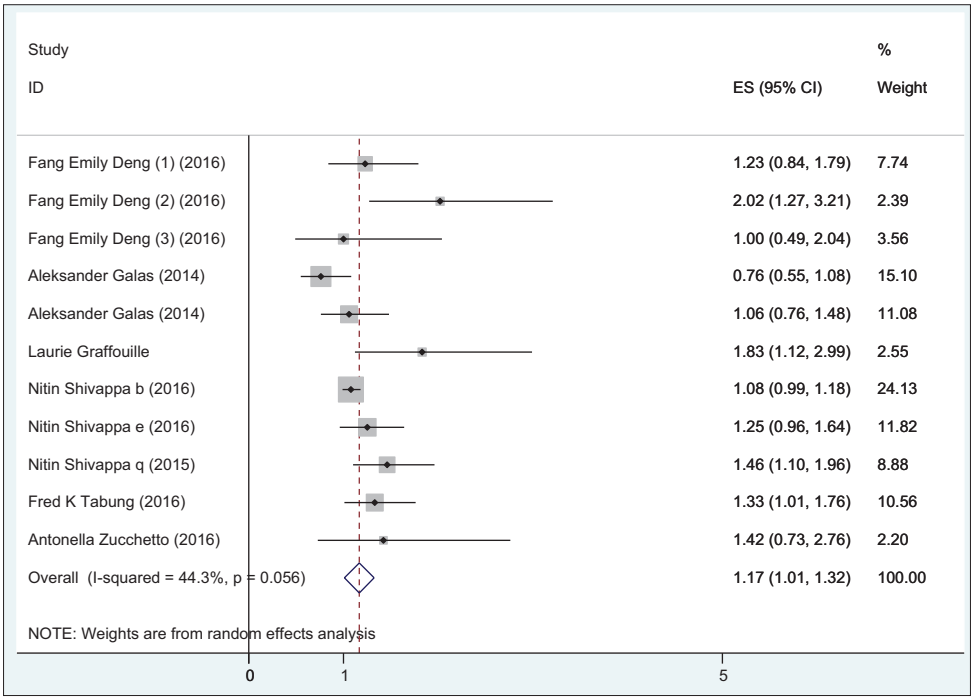

Figure 4: Odds ratio and 95% CI of individual studies and pooled data for the association between DII and mortality of cancer according to the type of cancer using random-effect model. OR: Odds of ratios

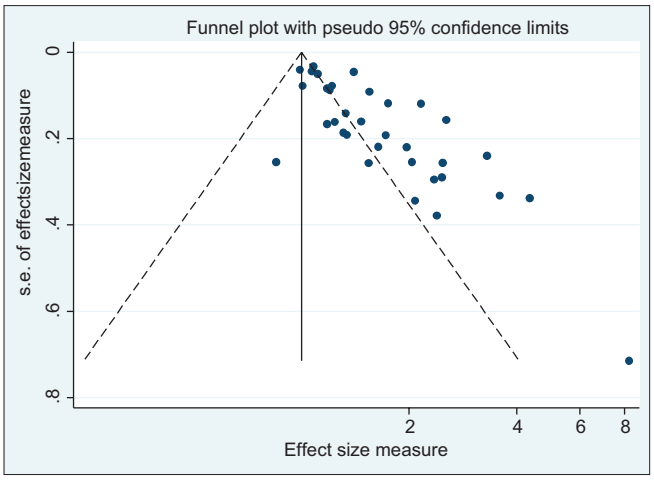

Figure 5: Funnel plot detailing publication bias in the studies reporting the association between DII and all cancer morbidity

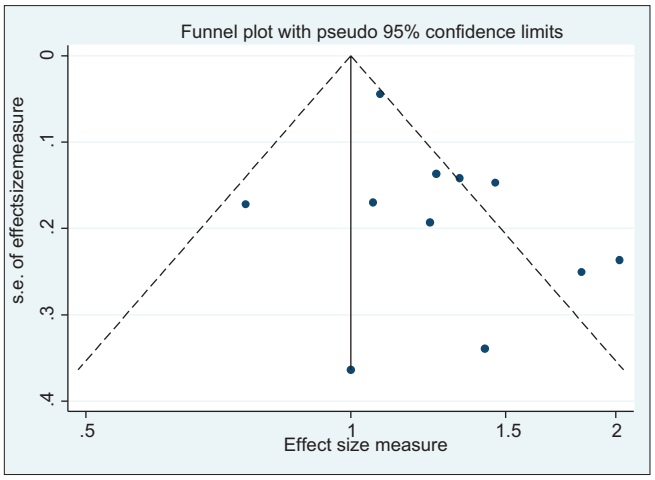

Figure 6: Funnel plot detailing publication bias in the studies reporting the association between DII and cancer mortality

literature by investigating the association between dietary components and inflammation. However, in contrast to the other dietary patterns, DII focuses on specific biological pathways modulating the impact of dietary factors on inflammation.[21] In fact, in comparison to other dietary pattern, DII can provide more comprehensive information on additional variables affecting inflammation.[25-29]

The present meta-analysis has some strengths and limitations. The main strength is that the study includes all indices of incidence, mortality, and length of hospitalization of cancers in relation with a categorical and continuous score of DII. In addition, we carried out the meta-analysis on all types of cancer. The limitations of the study were as follows: (a) reviewed studies were heterogeneous in terms of population characteristics, design, and duration of follow-up periods; and (b) the questionnaires used for food assessment were different. However, we tried to reduce the effect of heterogeneity on estimated effect sizes by using a random-effect model of analysis.

Conclusions

In conclusion, the present meta-analysis suggested a significant association between DII and incidence, mortality, and hospitalization in patients with different types of cancers. DII, which is used for evaluating inflammatory properties of diets, can be used as an appropriate tool to predict the incidence and mortality of all cancer types. According to the results of the study, we recommend that changing dietary patterns as malleable factors can substantially reduce both incidence and mortality risks in cancer patients.

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Conflicts of interest

There are no conflicts of interest.

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