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REVIEW

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Association between dietary inflammatory index and risk of chronic kidney disease and low glomerular filtration rate; a systematic review and meta-analysis of observational studies

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Abstract

Objective Earlier studies on the association between the dietary inflammatory index (DII) and the risk of chronic kidney disease (CKD) and low estimated glomerular filtration rate (low-eGFR) have provided uncertain findings. Therefore, this study aimed to summarize the existing literature on the association between DII and CKD and low-eGFR.

Methods In April 2024, PubMed, Scopus, and Web of Science were searched for observational studies, along with manual inclusion of Google Scholar and Embase. The review was submitted to PROSPERO (CRD42024536756) and conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement guidelines. Studies which reported risk for CKD or low-eGFR were included.

Results The random-effects model was used for statistical analysis and pooled effect sizes were reported as odds ratios (ORs) and their corresponding 95% confidence intervals (CIs). A total of 13 studies, all with a cross-sectional design, were identified eligible for inclusion in the meta-analysis. The results revealed that higher DII scores were associated with significantly higher odds of CKD (OR: 1.36, 95% CI: 1.20–1.56, $p < 0.001$) and low-eGFR (OR: 1.58, 95% CI: 1.26-2.00, $p = 0.001$).

Conclusion This study found a significant positive association between the DII and the odds of CKD and low-eGFR, suggesting a higher likelihood of CKD in individuals who adhere to a pro-inflammatory diet. Large-scale prospective cohort studies are required to confirm these findings, particularly by assessing different indicators of kidney function.

Keywords Dietary inflammatory index, Chronic kidney disease, Glomerular filtration rate, Inflammation

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Introduction

Chronic kidney disease (CKD) is a progressive condition that affects more than 800 million individuals worldwide. In contrast to cardiovascular and respiratory diseases, CKD mortality has been rising and reported as the third fastest-growing cause of death globally [1, 2]. It has been projected that by 2040, CKD will be the 5th highest cause of years of life lost globally [3]. Previous studies have suggested inflammatory processes as the pathogenesis of the majority of kidney diseases either in the development or progression [4–6]. Systemic or intrarenal inflammation may disrupt microvascular response to regulatory factors and promotes the production of various tubular toxins, such as reactive oxygen species. This process causes tubular injury, nephron dropout, and ultimately leads to the onset of CKD [7]. Additionally, elevated levels of systemic inflammatory markers are associated with a reduction in glomerular filtration rate (GFR) and an increase in the levels of urinary protein [8]. Therefore, reducing inflammation seems to be a potential strategy to prevent or reduce the progression of CKD [9, 10].

Among various risk factors proposed for CKD, dietary habits have been extensively explored in relation to renal function [11, 12]. Studies have suggested that anti-inflammatory dietary patterns such as Mediterranean diet are associated with lower inflammatory status [13–16]. These dietary patterns are mostly rich in plant-derived foods including vegetables, fruits, whole grains, nuts and seeds [17]. On the other hand, pro-inflammatory dietary patterns such as Westerns dietary pattern or high-glycemic index diets are associated with higher systemic inflammation [18–20]. Therefore, identifying a dietary pattern that holds the potential to reduce the risk of systemic inflammation might contribute to a more comprehensive prevention and treatment strategy for kidney diseases.

The dietary inflammatory index (DII) as a literature-derived population-based index for assessing the potential inflammatory effects of diets [21], has been investigated in relation to the risk of CKD or low estimated GFR (low-eGFR) by several studies [22–25]. While several of these studies have reported a significant increase in the risk of CKD and low-eGFR in participants who adhered to a pro-inflammatory diet (high DII score) [24, 26, 27], the reported effect sizes varied, with differences in the strength of associations and the specific odds ratios presented.

Therefore, since no previous systematic review or meta-analysis has comprehensively evaluated the relationship between DII and risk of CKD or low-eGFR, this study aimed to summarize the existing literature and clarify the association between DII and the risk of CKD and low-eGFR, as well as its relationship with serum biomarkers of kidney function. It is important to note that while individuals with low-eGFR may be classified as CKD patients, CKD is identified by the presence of albuminuria and/or low-eGFR. This distinction indicates that the risk of CKD in a specific population is not necessarily the same as the risk of low-eGFR [28]. Consequently, our study aimed to evaluate the risk of CKD and low-eGFR separately.

Methods

Study design

We systematically reviewed the relationship between DII and risk of CKD or low-eGFR as well as its correlation with uric acid, blood urea nitrogen (BUN), and creatinine. The review was submitted to PROSPERO (CRD42024536756) and conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement guidelines.

Search strategy

In April 2024, a comprehensive search was conducted using Medical Subject Heading (MeSH) terms and text words related to DII, CKD, and low-eGFR combined with relevant renal function parameters (Supplementary Table 1). This search was updated weekly until August 2024. PubMed, Scopus, and Web of Science Core Collection databases were searched without any restrictions on publication date or language. Additionally, a manual search was conducted to identify any remaining articles; however, no additional records were included through this method. Figure 1 shows the search strategy and the total number of studies evaluated and selected.

Selection criteria

Two independent reviewers (AAK and FK) screened titles and abstracts to find relevant articles. No relevant articles in languages other than English were identified. Disagreements were resolved with the assistance of FH. Based on the full text article and the inclusion and exclusion criteria, eligible studies were identified. The studies were identified as eligible if they met the following criteria: [1] original studies on adult population (aged 18 or older) [2], an observational design (cross-sectional, prospective cohort, or case-control) [3], reporting risk assessed by odds ratio (OR) or relative risk (RR) or hazard ratio (HR) with their corresponding 95% confidence intervals (CIs) for low-eGFR (defined as eGFR below 60%) or CKD (defined as presence of albuminuria and/or low-eGFR) [84–86] (Table 1) [4] or reporting any type of association between DII and serum biomarkers of renal function or eGFR [28, 29]. Studies were excluded if they were not original research, were in vitro or animal model, conducted on children, or did not have outcomes of interest.

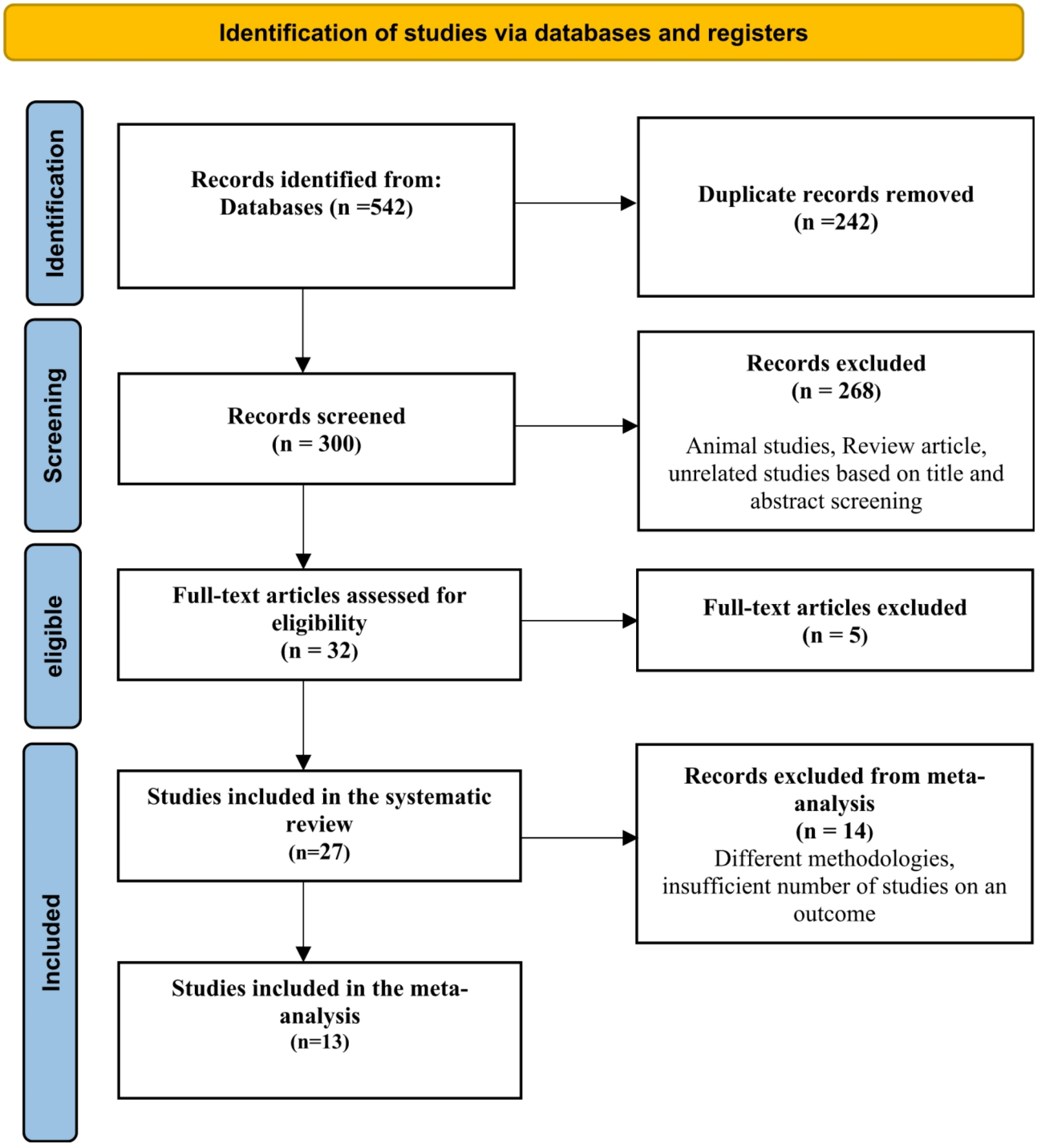

Fig. 1 Flow diagram of study selectin

Table 1 Definitions of CKD and Low-eGFR

Outcome	Definition	Notes
Low-eGFR	eGFR < 60 mL/min/1.73 m^2 (29)	Low-eGFR categorized as stages G3–G5 per KDIGO guidelines (29). GFR Calculated using the CKD-EPI or MDRD formula [84, 85]
CKD	Presence of albuminuria and/or low-eGFR (29, 86)	Albuminuria defined as ACR ≥ 30 mg/g [86]

Abbreviation: eGFR, estimated glomerular filtration rate; CKD, chronic kidney disease; CKD-EPI, chronic kidney disease epidemiology collaboration; MDRD, modification of diet in renal disease; ACR, albumin-to-creatinine ratio; KDIGO, kidney disease: improving global outcomes

Data extraction

If there were several articles related to a single study, we prioritized the latest publication. The following data were extracted from the studies: first author's name, publication year, country, design, dietary assessment method, participant's health status, sample sizes, outcomes, final results, and adjusted variables.

Quality assessment

The quality of studies was assessed by two authors (AAK and FK) independently, utilizing the Newcastle-Ottawa scale for cross-sectional and cohort studies (Supplementary Table 3) [30]. Additionally, the Risk of Bias in Non-Randomized Studies of Environmental Exposures (ROBINS-E) tool was used to assess the risk of bias and the quality of the included cohort studies (Supplementary Table 4) [31]. Disagreements were resolved with the assistance of FH.

Certainty assessment

The overall certainty of evidence across the studies was assessed using the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) guidelines. The quality of evidence was classified into four categories, based off the corresponding evaluation criteria: high, moderate, low, and very low.

Statistical analysis

The articles which reported the association between high DII and risk of CKD or low-eGFR in comparison with low DII using OR, RR, and HR were included in the current meta-analysis. If RRs or HRs were reported, they were treated as equivalent to ORs when the prevalence of frailty in the study population was ≥ 20% [32]. To summarize the association between high DII and CKD or low-eGFR, risk estimates extracted from each study were calculated using the average of the natural logarithm ORs. We used the random-effects model and the inverse-variance method to calculate the pooled effect size. Heterogeneity was evaluated using the I^2 statistic [33]. To explore potential sources of heterogeneity, we conducted subgroup analyses based on dietary assessment tool (24-hour recall/Food Frequency Questionnaire), the methods used to calculate the DII (DII/E-DII), the participants sex (both sexes/females only), and geographical regions (United States/Asia). Publication bias was examined using visual inspection of a funnel plot, Egger's, and Begg's tests. When bias was detected, a trim-and-fill analysis was performed to assess its impact on the overall effect. Additionally, a sensitivity analysis was conducted to determine the impact of each study on the pooled effect by removing any specific study. All analyses were conducted using Stata 11.0 software (StataCorp, College Station, TX), and significance was set at p values < 0.05.

Results

Study selection and characteristics

The search strategy (Supplementary Table 1) yielded 537 records. After removing 242 duplicates, an initial screening of 300 studies based on their titles and abstracts resulted in the exclusion of 268 records. After conducting eligibility assessments on 32 full texts, 27 studies met the criteria to be included in the systematic review. Among them, 24 were cross-sectional studies [16, 22–27, 34–49], one was case-control study [50], and two were cohort studies [51, 52], one of which included both cross-sectional and cohort data [52]. Furthermore, among the included studies, 13 were eligible for the quantitative synthesis (meta-analysis). The included studies in the systematic review were published between 2015 and 2024, with a sample size ranging from 150 to 66,978. In total, the included studies involved 290,890 participants. Detailed characteristics of the included studies are shown in Table 2. The review included studies conducted in various countries: 11 in North America [16, 24, 36–39, 46–49, 53], 9 in Asia [23, 26, 27, 35, 40, 42, 43, 45], 7 in Europe [22, 25, 41, 44, 50, 52], and one in Australia [52]. Twelve articles examined the association between DII and CKD or diabetes kidney disease (DKD) [16, 26, 27, 34, 36, 37, 39, 42, 46, 48, 49, 53], while 14 studies reported the association between DII and GFR [16, 24–26, 35–42, 47, 52]. Furthermore, 4 studies reported the association between DII and serum uric acid [33, 38–40], 8 studies reported creatinine [22, 23, 40, 42–45, 50], and 3 studies reported BUN [23, 42, 43].

Quality assessment

Based on the Newcastle-Ottawa Scale, the current systematic review included two cohort studies by Li et al. and Bondonno et al., which received scores of 6 and 7 out of nine, respectively [51, 52], as well as one case-control study that received a score of 6 [50]. However, the scores of the cross-sectional studies varied more widely (Supplementary Table 3). The quality evaluation results of the included articles ranged from 3 [45] to 7 out of 9 [26, 27, 35, 38, 39, 41, 48, 52]. Most studies did not report non-response rates and were not scored on this criterion. Nevertheless, the majority of the studies received moderate to good quality assessments. Furthermore, based on the ROBINS-E tool, the two included cohort studies had a moderate to serious risk of bias (Supplementary Table 4) [51, 52].

Certainty assessment

To assess the quality of the evidence for main outcomes in this systematic review and meta-analysis, the GRADE framework was performed (Supplementary Table 5). The results indicated an overall low certainty regarding the association between DII and the risk of CKD and low

Table 2 General characteristics of included studies in the systematic review and meta-analysis

Author Year Country	Study design	method of Dietary assessment	Population (Health status, *N*, sex, mean age)	Outcomes	Results	Adjusted variables	Quality score
Huang J 2024 USA [24]	cross-sectional	24 h-Recall	General population *N* = 2108 M = 1044 F = 1064 Mean age: 53.9	1. Risk of low-eGFR 2. Correlation with eGFR	1. Highest vs. lowest adherence: OR = 2.070; 95%CI, 1.12, 3.82 2. Significant negative correlation between DII and eGFR.	Gender, age, race, education level, poverty index, hypertension, diabetes, smoking, and drinking.	6/9
Guo M 2024 China [26]	cross-sectional	24 h-Recall	Middle-aged and elderly populations *N* = 23,175 M=- F=- Mean age: 60	1. Risk of CKD 2. Risk of low-eGFR	1. Highest vs. lowest adherence: OR = 1.08; 95%CI, 1.05, 1.10 2. Highest vs. lowest adherence: OR = 1.16; 95%CI, 1.13, 1.19	Race, age, sex, education level, smoking status, PIR, BMI, albumin, glucose, glycosylated hemoglobin, ALT, AST, serum iron, potassium, cholesterol, triglycerides, BUN, uric acid, hypertension, coronary heart disease, congestive heart failure, stroke, arthritis, cancer, and diabetes.	7/9
Guo C 2023 China [27]	cross-sectional	24 h-Recall	Patients with type-2 diabetes mellitus *N* = 7974 M = 4131 F = 3843 Mean age: 59.5	Risk of CKD	Highest vs. lowest adherence: OR = 1.67; 95%CI, 1.29, 2.17	Age, sex, ethnicity, smoking, drinking, body mass index (BMI), triglyceride (TG), total cholesterol (TC), metabolic equivalents (METs), energy intake, hypoglycemic medications, hypertension, and cardiovascular disease (CVD).	7/9
Moludi 2022 Iran [34]	cross-sectional	FFQ	General population *N* = 9824 M = 4610 F = 5214 Age: 35–65	Risk of CKD	Highest vs. lowest adherence: OR = 1.92; 95%CI, 1.52–2.42	Age, gender, smoking status, BMI, place, education level, and physical activity, HEI, kidney stone, diabetes, and high blood pressure.	5/9
Lin 2021 China [35]	cross-sectional	FFQ	Women with diabetes, prediabetes and normal glucose *N* = 2644 F = 2644 Mean age: 55.3	1. Risk of low-eGFR 2. Correlation with eGFR	1a. Highest vs. lowest adherence in diabetes: OR = 15.519; 95%CI, 1.373, 175.377 1b. Highest vs. lowest adherence in prediabetes: OR = 2.413; 95%CI, 0.688, 8.461 1c. Highest vs. lowest adherence in normal blood glucose participants: OR = 1.439; 95%CI, 0.250, 8.300 2. Significant negative correlation between DII and eGFR in patients with diabetes. But no significant correlation in patients with pre-diabetes and normal glucose.	Age, BMI, current smoking, alcohol intake and exercise, systolic BP, diastolic BP, glucose, LD, HDL, triglyceride, cholesterol	7/9

Table 2 (continued)

Author Year Country	Study design	method of Dietary assessment	Population (Health status, *N*, sex, mean age)	Outcomes	Results	Adjusted variables	Quality score
Mazidi 2018 USA [16]	cross-sectional	24 h-Recall	General population *N* = 21,649 M= - F= - Age: ≥18	1. Risk of CKD 2. Risk of low-eGFR 3. Association with eGFR 4. Association with Cr 5. Association with uric acid	1. Highest vs. lowest adherence: OR = 1·23; 95%CI, 1.10, 1.35 2. Highest vs. lowest adherence: OR = 1·29; 95%CI, 1·03, 1·62 3. Significant decrease in mean eGFR across increasing E-DII quartiles. 4. Nonsignificant increase in mean Cr across increasing E-DII quartiles. 5. Significant decrease in mean uric acid across increasing E-DII quartiles.	Blood glucose, blood pressure, BMI, diabetes, hypertension status	6/9
Wang 2022 USA [36]	cross-sectional	24 h-Recall	Diabetes *N* = 4264 M = 2241 F = 2023 Age: >20	1. Risk of low-eGFR 2. Risk of DKD	1. Highest vs. lowest adherence: OR = 1.57; 95%CI, 1.10, 2.26 2- Highest vs. lowest adherence: OR = 1.64; 95%CI, 1.24, 2.17	Age, sex, race, educational level, marriage status, family poverty income ratio, smoking status, drinking status, physical activity level, hypertension and BMI.	6/9
Qu 2024 USA [37]	cross-sectional	24-hour dietary recall	General population *N* = 18,070 M = 8906 F = 9164 Age: ≥ 20	1. Risk of CKD 2. Risk of low-eGFR	1. Highest vs. lowest adherence: OR = 1.24; 95%CI, 1.11, 2.64 2. Highest vs. lowest adherence: OR = 1.71; 95%CI, 0.95, 1.63	Age, sex, race, education level, marital status, PIR, BMI, smoking status, physical activity, diabetes, hypertension and energy	6/9
Zeng 2023 USA [38]	cross-sectional	24-hour dietary recall	Adults Aged 50 Years and Older *N* = 12,090 M= - F= - Age: ≥ 50	Risk of low-eGFR	Highest vs. reference adherence: OR = 2.08; 95%CI, 1.30–2.86	Age, sex, body mass index, race/ethnicity, education, ratio of family income to poverty, smoking, physical activity, hypertension, diabetes, and daily intakes of total plain water, total energy, and sodium.	7/9

Table 2 (continued)

Author Year Country	Study design	method of Dietary assessment	Population (Health status, *N*, sex, mean age)	Outcomes	Results	Adjusted variables	Quality score
Xu Z 2024 USA [39]	cross-sectional	24 h-Recall	CKD patients *N* = 2488 M: 1183 F: 1304 Mean age: 67.11 ± 15.56	1. Risk of higher CKD Stages 2. Correlation with eGFR	1- Highest vs. reference adherence: OR = 2.29; 95%CI, 1.42, 3.71 2. Significant negative correlation between DII and eGFR.	Age, gender, race, education level, poverty income ratio (PIR), marital status, body mass index (BMI), metabolic equivalent (MET) score, drinking, smoking, history of hypertension, history of diabetes, cotinine, systolic blood pressure, diastolic blood pressure, total triglycerides, and total cholesterol	7/9
Vahid 2023 Luxemburg [25]	cross-sectional	FFQ	General population *N* = 1404 M = 654 F = 750 Age: 25–79	1. Correlation with eGFR 2. Correlation with uric acid	1. No Significant correlation between DII and eGFR. 2. No Significant correlation between DII and uric acid.	Age, gender, birth country, marital status, education, job, income, IPAQ scoring, current smoking	6/9
Bondonno 2020 Australia [52]	Cohort (10 years)/ cross-sectional	FFQ	Older women *N* = 2644 F = 2644 Mean age: 55.3	Correlation with eGFR	Significant negative correlation between DII and eGFR both at the baseline and after 10 years.	Age, energy intake, treatment code (calcium or placebo), BMI, smoking status, physical activity, alcohol intake, diabetes status, use of antihypertensive medication, prevalent ASVD, statin use, and use of NSAIDs for joint pain	7/9
Tajik 2019 Iran [40]	cross-sectional	FFQ	Elderly population *N* = 221 M = 65 F = 161 Mean age: 67 ± 5.7	1. Correlation with eGFR 2. Association with Creatinine	1. No Significant correlation between DII and eGFR. 2. No significant association between mean Cr and DII.	Energy intake, age, sex, BMI, smoking status, physical activity, hypertension, diabetes, use of lipid-lowering medication, angiotensin II receptor blockers (ARB) and angiotensin-converting enzyme inhibitor (ACEI), steroidal and non- steroidal anti-inflammatory medications.	6/9
Xu H 2015 Sweden [41]	cross-sectional	7-d food records	Elderly population *N* = 1942 M = 1520 F = 422 Age: 70–71	Correlation with eGFR	Significant negative correlation between DII and eGFR.	Energy intake, age, sex, smoking status, physical activity, hypertension, diabetes, use of lipid-lowering medication, and whether the participants were from the Uppsala Longitudinal Study of Adult Men or the Prospective Investigation of Vasculature in Uppsala Seniors, BMI, CRP	7/9

Table 2 (continued)

Author Year Country	Study design	method of Dietary assessment	Population (Health status, *N*, sex, mean age)	Outcomes	Results	Adjusted variables	Quality score
Rouhani 2018 Iran [42]	cross-sectional	FFQ	CKD patients *N* = 221 M= - F= - Mean age: -	1. Risk of higher CKD Stages 2. Association with eGFR 3. Association with Cr 4. Association with BUN	1. Highest vs. lowest adherence: OR = 2.12; 95%CI, 1.05, 4.26 2. No significant association between mean eGFR and DII. 3. No significant association between mean Cr and DII. 4. No significant association between mean BUN and DII.	Socioeconomic status, height and weight, systolic and diastolic blood pressure	5/9
Alkerwi 2015 Luxembourg [22]	cross-sectional	SQ-FFQ	General population *N* = 1352 M= - F= - Age: 18–69	1. Correlation with Cr 2. Correlation with uric acid	1. No Significant correlation between DII and Cr 2. No Significant correlation between DII and uric acid.	Age (continuous), sex, education level (primary, secondary or tertiary), smoking status (smoker or non-smoker), physical activity in metabolic equivalents-h/week	5/9
Farhangi 2018 Iran [23]	cross-sectional	SQ-FFQ	Candidates of CABG surgery *N* = 454 M = 332 F = 122 Age: 35–80	1. Correlation with Cr 2.Correlation with BUN	1. Significant positive correlation between DII and Cr in male patients, but not in female patients. 2. Significant positive correlation between DII and BUN in male patients, but not in female patients.	Age, gender, BMI, educational attainment and presence of diabetes and myocardial infarction	5/9
Bavi Behbahani 2022 Iran [43]	cross-sectional	FFQ	Atherosclerosis patients *N* = 320 M = 171 F = 149 Age: ≥ 20	1. Correlation with Cr 2. Correlation with BUN	1. No Significant correlation between DII and Cr. 2. No Significant correlation between DII and BUN.	Age, sex, energy intake, physical activity, race, BMI, WC, marital status, and education.	5/9
Rodgers 2024 Spain [50]	Case-control	SQ-FFQ	Cases: kidney stone formers. Controls: individuals with no history of kidney stone. *N* = 160 (Cases:97, Controls:63) M = 111 F = 49 Mean Age: 47	1. Correlation with Cr 2. Correlation with uric acid	1. No Significant correlation between DII and Cr. 2. No Significant correlation between DII and uric acid.	Sex, age and for the statistically significant predictors of the univariate analyses	6/9
Carrasco-Marín 2024 UK [44]	cross-sectional	24 h dietary recall	Healthy adults *N* = 66,978 M = 30,852 F = 36,126 Age: 37–73	1. Correlation with Cr 2. Correlation with uric acid	1. Significant negative correlation between DII and Cr. 2. No Significant correlation between DII and uric acid.	Age, sex, deprivation, smoking, alcohol consumption, physical activity, and BMI.	6/9

Table 2 (continued)

Author Year Country	Study design	method of Dietary assessment	Population (Health status, *N*, sex, mean age)	Outcomes	Results	Adjusted variables	Quality score
Kizil 2016 Turkey [45]	cross-sectional	3-day dietary recall	Hemodialysis patients *N* = 150 M = 68 F = 82 Mean age: 57.5 ± 12.4	Association with Cr	No significant association between mean Cr and DII.	Gender, education level, and marital status.	3/9
Lu 2024 USA [46]	cross-sectional	24 h-Recall	patients with hypertension *N* = 17,294 M = 8642 F = 8652 Mean age = 59.78 ± 0.18	Risk of CKD	Highest vs. lowest adherence: OR = 1.38; 95%CI, 1.15,1.65	Age, gender, race, education, family income, smoking status, alcohol intake, and diabetes.	6/9
Rivera-Paredez 2024 Mexico [47]	cross-sectional	SQ-FFQ	patients with hypertension *N* = 2098 M = 32.4% F = 67.6% Mean age = 47	Correlation with eGFR	Significant negative correlation between DII and eGFR.	Age, smoking, drinking, physical activity, hypertension, BMI, glucose, lipids, and blood pressure	6/9
Rui 2024 USA [48]	cross-sectional	24 h-Recall	patients with DM *N* = 2712 M = 32.4% F = 67.6% Mean age = 47	Risk of DKD	Highest vs. lowest adherence: OR = 0.9; 95%CI, -0.6, 2.4	Age, gender, race, education level, PIR, BMI, waistline, total energy intake, HbA1c, FPG, Ualb, Ucr, BUN, SUA, TC, TG, HDL, LDL, ALT, AST, Hypertension, MetS, taking prescription for hypertension, taking prescription for to lower blood sugar, Taking prescription for cholesterol	7/9
Huang Y 2024 USA [53]	cross-sectional	24 h-Recall	General population *N* = 25,167 M = 48.3% F = 51.7% Mean age = 49.2	Risk of CKD	Highest vs. lowest adherence: OR = 1.56; 95%CI, 1.34, 1.82	Age, gender, race/ethnicity, body mass index, smoking status, poverty status, education levels, alcohol consumption, leisure time physical activity, history of diabetes.	6/9

Table 2 (continued)

Author Year Country	Study design	method of Dietary assessment	Population (Health status, *N*, sex, mean age)	Outcomes	Results	Adjusted variables	Quality score
Guo L 2024 USA [49]	cross-sectional	24 h-Recall	General population *N* = 40,388 Without CKD: M = 50.42% F = 49.58% Mean age = 44.99 With CKD: M = 43.20% F = 56.80% Mean age = 60.84	Risk of CKD	Highest vs. lowest adherence: OR = 1.24; 95%CI, 1.12, 1.37	Sex, age group, race, education, marital status, BMI, smoking, and drinking status, hypertension, and diabetes.	6/9
Li 2024 USA [51]	Cohort (19 years)	24 h-Recall	General population *N* = 23,099 M= - F= - Age = ≥ 18	Risk of CKD	Highest vs. lowest adherence: HR = 1.36; 95%CI, 1.23–1.51	Systolic blood pressure (SBP), total cholesterol (TC), age, education, BMI, serum creatinine, smoking habit, sex, and race.	6/9

Abbreviation: FFQ, food frequency questionnaire; DII, dietary inflammatory index; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; E-DII, estimated dietary inflammatory index; OR, odds ratio; HR, hazard ratio; CI, confidence interval; Cr, creatinine; BUN, blood urea nitrogen; DKD, diabetic kidney disease

eGFR, as well as the correlation between DII and eGFR. Additionally, very low certainty was found for the relationships between DII and creatinine, uric acid, and BUN.

Findings

Table 2 summarizes the systematic review results, providing details on study design, setting, population, outcomes, and findings.

Meta-analysis findings

DII and CKD risk

A total of nine studies, comprising 169,346 participants, investigated the association between DII and the risk of CKD, with all reporting a significantly higher risk of CKD in participants with the highest DII scores [16, 26, 27, 34, 37, 46, 49, 51, 53]. The results of our meta-analysis revealed 36% higher odds of CKD in participants with the highest DII scores compared to those with the lowest (95% CI: 1.20–1.56, $p < 0.001$) (Fig. 2). Significant heterogeneity was observed between studies ($I^2 = 90.4\%$; $P < 0.001$). When studies were stratified by the dietary assessment tool, the methods used to calculate the DII, and the geographical region where studies conducted, results in the subgroups remained consistent with the overall estimate, and heterogeneity did not disappear (Table 3). There was evidence of publication bias, as suggested by an asymmetry in the funnel plot and the Egger test ($P < 0.001$). Additionally, the trim and fill algorithm indicated an adjusted value, showing a direct association between the DII and the odds of CKD (OR: 1.363, 95% CI: 1.208 to 1.537). The slight difference between the adjusted value (1.363) and the original estimate (1.360) suggests a minor influence of the study effect on the original results. Sensitivity analysis consistently supported a positive association between DII and CKD risk, indicating the robustness of this relationship.

DII and risk of low e-GFR

In total, 7 studies with 84,000 participants investigated the association between DII and risk of low-eGFR [16, 24, 26, 35–38]. All of these studies reported a significantly higher odds of low-eGFR in participants with the highest adherence to DII, compared to those with the lowest adherence. Our meta-analysis revealed that high DII scores were associated with a 58% increase in the odds of low eGFR (95% CI: 1.26-2.00, $p = 0.001$). (Fig. 3). The studies showed significant heterogeneity ($I^2 = 70\%$; $P = 0.001$). When studies were stratified by the dietary assessment tool, the methods used to calculate the DII, participants sex, and the study geographical region, results in the subgroups remained consistent with the overall estimate and in some cases, the heterogeneity level decreased considerably (Table 4). Specifically,

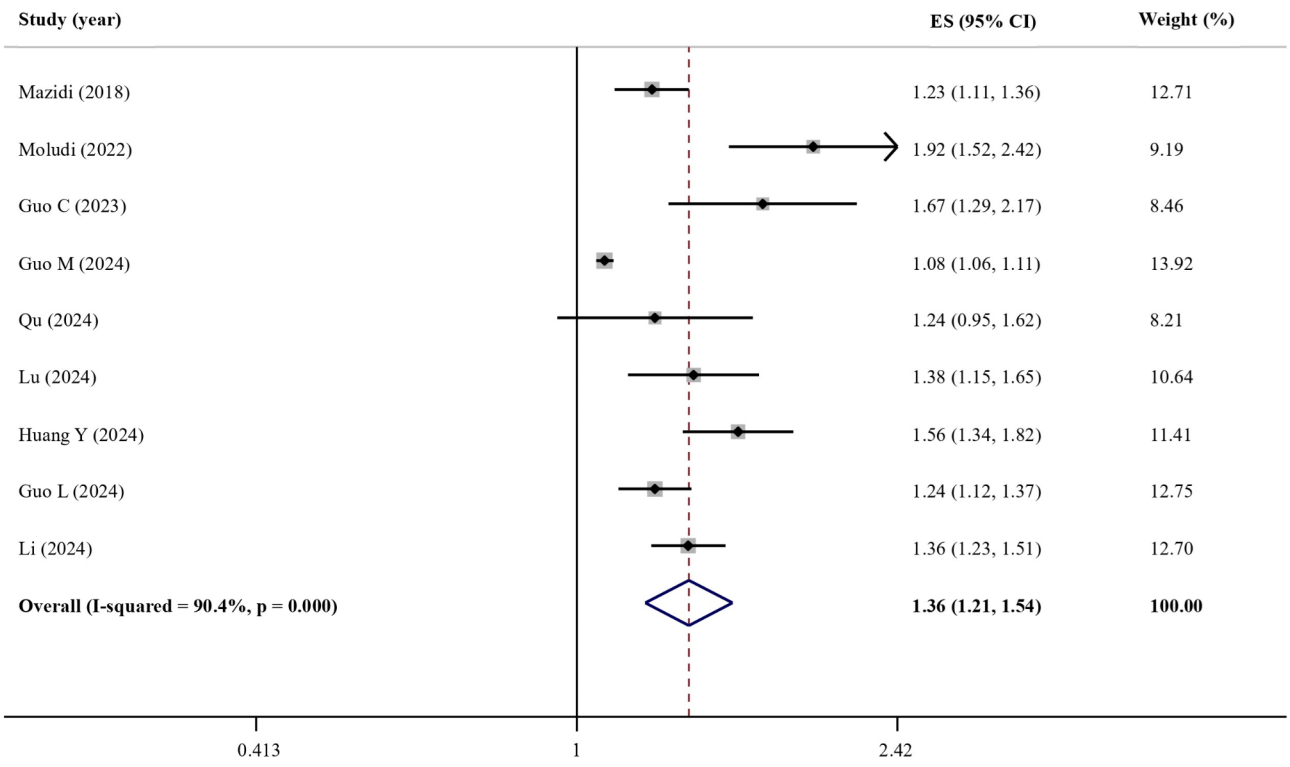

Fig. 2 Forest plot of studies investigating the association of dietary inflammatory index with chronic kidney disease risk

Table 3 Subgroup analysis for the association between DII and CKD risk

Subgroup	Effect size	I^2	OR	95%CI	*P*between
Dietary intake assessment					< 0.001
24 h-Recall	8	88.9%	1.31	1.17–1.47	
FFQ	1	-	1.92	1.52–2.42	
Country					< 0.001
US	8	88.9%	1.31	1.17–1.47	
Asia	1	-	1.92	1.52–2.42	
Type of DII					< 0.001
DII	8	91.3%	1.39	1.21–1.60	
E-DII	1	-	1.23	1.11–1.36	

Abbreviation: FFQ, food frequency questionnaire; DII, dietary inflammatory index; CKD, chronic kidney disease; E-DII, estimated dietary inflammatory index

heterogeneity decreased significantly in studies that used food frequency questionnaires (FFQ) as the dietary assessment tool ($I^2 = 20.7\%$; $P = 0.283$), were conducted in Asia ($I^2 = 20.7\%$; $P = 0.283$), used the energy-adjusted DII (E-DII) ($I^2 = 38.5\%$; $P = 0.181$), and enrolled only women ($I^2 = 20.7\%$; $P = 0.283$). An asymmetry in the funnel plot and the Egger test ($P = 0.002$) suggested possible publication bias. Additionally, according to the trim and fill algorithm, the adjusted value indicated a direct association between the DII and the odds of low-eGFR (OR: 1.704, 95% CI: 0.878 to 2.530). Comparing the adjusted value (1.704) with the original estimate (1.58) suggests a small contribution of the study effect to the original results. Sensitivity analysis revealed a consistent positive link between DII and the odds of low-eGFR by excluding each individual study, indicating the robustness of our results.

Narrative review

DII and risk of CKD progression

In total, 2 studies used a cross-sectional design to investigate the relationship between DII and the odds of CKD progression [39, 42]. These studies reported the OR for being in the higher stages of CKD, which reflects disease progression rather than the overall odds of CKD. Consequently, we excluded these studies from the meta-analysis. Both studies reported an increased odds of CKD progression in participants with highest DII scores. Rouhani et al. reported an increased odds of being in the higher stages of CKD among those in the top tertiles of

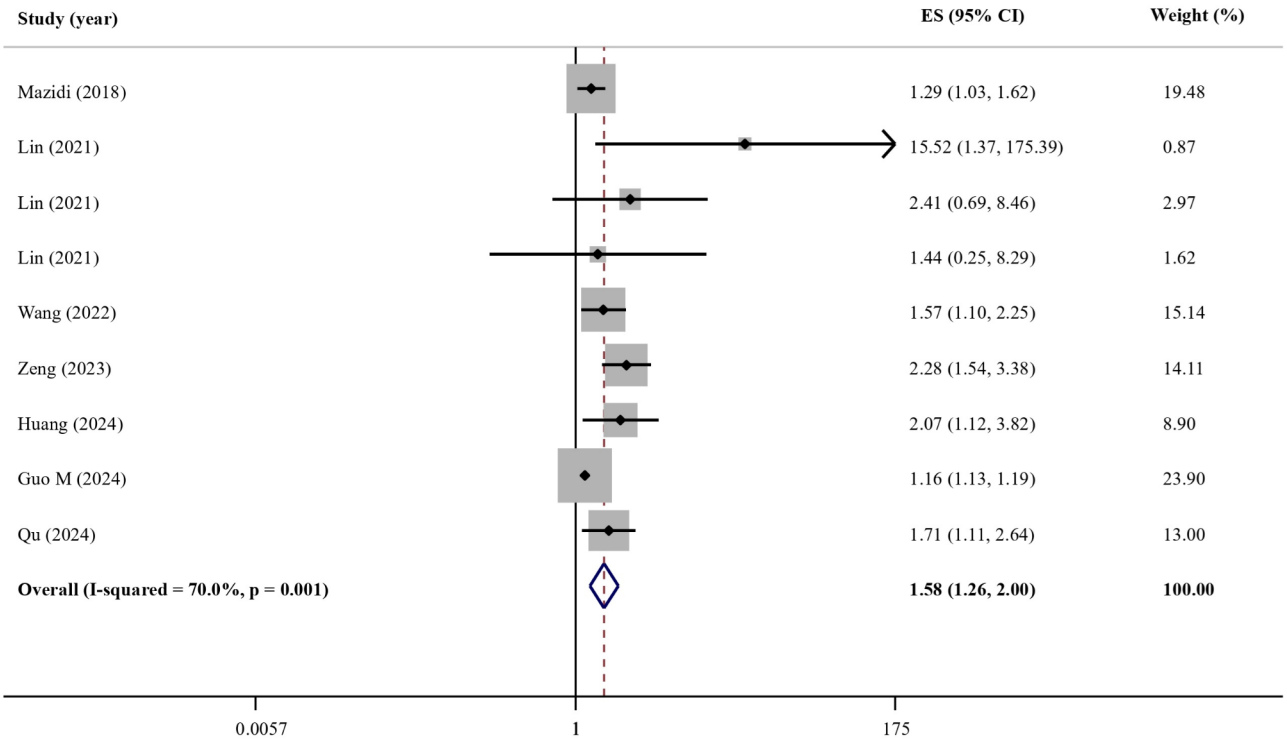

Fig. 3 Forest plot of studies investigating the association of dietary inflammatory index with low estimated glomerular filtration rate risk

Table 4 Subgroup analysis for the association between DII and risk of low-eGFR

Subgroup	Effect size	I^2	OR	95%CI	*P*between
Dietary intake assessment					0.001
24 h-Recall	6	76.2%	1.52	1.22–1.91	
FFQ	3	20.7%	2.87	0.96–8.61	
Country					0.001
US	6	76.2%	1.52	1.22–1.91	
Asia	3	20.7%	2.87	0.96–8.61	
Type of DII					0.001
DII	8	80.2%	1.64	1.19–2.26	
E-DII	1	38.5%	1.82	0.88–3.75	
Sex					0.001
Both	6	76.2%	1.52	1.22–1.91	
Female	3	20.7%	2.87	0.96–8.61	

Abbreviation: FFQ, food frequency questionnaire; DII, dietary inflammatory index; eGFR, estimated glomerular filtration rate; E-DII, estimated dietary inflammatory index

DII compared to those in tertile 1 (OR = 2.12, 95% CI: 1.05–4.26, $P = 0.03$) [42]. Xu et al. also found a positive association between DII and the odds of higher CKD stages (Q4 vs. Q1, OR = 2.29, 95% CI: 1.42–3.71, P for trend = 0.0007) [39].

DII and risk of DKD

Two studies explored the association between DII and diabetic kidney disease (DKD), yielding inconsistent results [36, 48]. Due to differences in the definitions of DKD and CKD, these studies were not included in the meta-analysis [28, 54]. Wang's study suggested that higher quartiles of DII were linked to an increased odds of DKD (Q4 to Q1, OR = 1.64, 95% CI: 1.24–2.17, $p < 0.05$) [36]. However, Rui's study did not show significant association between DII and DKD in the fully adjusted model [48].

Correlation between DII and eGFR

Ten studies examined the correlation between DII and eGFR [16, 24, 25, 35, 39–42, 47, 52]. Except for the Bondonno et al. cohort study [52], all the studies had a cross-sectional design. Seven studies indicated that higher DII was linked to a decrease in eGFR [16, 24, 35, 39, 41, 47,

52], while 3 studies did not observe any significant correlation [25, 40, 42].

DII and creatinine

Eight studies were conducted to evaluate the link between DII and creatinine [22, 23, 40, 42–45, 50]. All of the studies were cross-sectional, except for one, which was a case-control study [50]. While the majority of studies found no significant correlation between DII and creatinine [22, 40, 43, 45, 50], 2 studies reported significant associations [23, 44]. Carrasco-Marín et al. reported that individuals with a pro-inflammatory diet had significantly higher levels of creatinine (β = 0.27, 95% CI 0.26–0.29, p-value < 0.0001) [44]. Similarly, a cross-sectional study by Farhangi et al. indicated that DII was linked to increased creatinine levels, specifically in men [23].

DII and uric acid

Four studies explored the link between DII and uric acid levels [22, 25, 44, 50]. All the studies employed a cross-sectional design except for a case-control study [50]. While 3 studies found no significant association between DII and uric acid [22, 25, 50], the study by Carrasco-Marín et al. indicated that individuals with a pro-inflammatory diet exhibited elevated uric acid levels (β = 0.21, 95% CI 0.29 – 0.23, p-value < 0.0001) [44].

DII and BUN

Three studies, all with a cross-sectional design, investigated the association between DII and BUN [42, 43]. The majority of studies did not find a significant association between DII and BUN [23, 42, 43]. The only study that found a significant association between DII and BUN was the study by Farhangi et al., which reported a direct correlation between them (Q4 compared to Q1, β = 1.04, 95% CI 1.01–1.08, p < 0.05) [23].

Discussion

The results of the present meta-analysis suggest that individuals with higher DII scores had 36% and 58% higher odds of CKD and low-eGFR, respectively. These associations were independent of the dietary assessment tool, the methods used to calculate the DII, participants sex, and the geographical region. Additionally, although the systematic review found a negative correlation between DII and eGFR, the correlation between DII and serum biomarkers of kidney function were inconsistent.

The association between pro-inflammatory dietary patterns and risk of developing different health condition has been extensively explored [55–57]. In 2021 Marx et al. conducted an umbrella review exploring the association between DII and different health conditions [57]. The study included 15 meta-analyses with a total population of 4,360,111 participants reporting 38 chronic disease-related outcomes. Marx et al. reported a significant positive association between adherence to a pro-inflammatory dietary pattern and 27 (71%) health outcomes such as myocardial infraction, all-cause mortality, and overall risk of cancer incidence [57]. However, due to the lack of any meta-analysis on DII and renal dysfunction at that time, Marx et al. failed to provide any information on CKD in their umbrella review [57].

In agreement with our findings, a recent meta-analysis reported a significant association between elevated risk of CKD and high DII scores [58]. However, the study had some limitations [87]. Firstly, Chen et al. included 3 cohort studies which examined the relationship between DII and mortality but not CKD development. Secondly, their search strategy has not been updated since March 2023 up until August 2024, leading to missing 7 relevant studies [26, 27, 37, 46, 49, 51, 53]. Thirdly, they combined the risk of CKD progression with other studies examining the risk of CKD [42]. For example, the study by Rouhani et al. was conducted on CKD patients and reported the OR for being in the higher stages of chronic kidney disease, which should not be interpreted as the risk of CKD development [42]. Finally, Chen et al. only explored the relationship between DII and CKD, while in the current study, the correlation between DII and different measures of kidney function, like GFR, creatinine, and BUN and risk of low-eGFR, has been investigated.

In line with our findings suggesting a positive association between DII and the risk of both CKD and low-eGFR, previous studies have reported an increased risk of CKD in individuals who adhered to pro-inflammatory dietary patterns, such as Western dietary pattern and diets high in ultra-processed foods [18, 59]. Furthermore, several studies have reported a negative association between anti-inflammatory dietary patterns and risk of CKD [15, 60]. For example, a meta-analysis by Hansrivijit et al. explored the association between CKD and the adherence to the Mediterranean Diet (MD), which has anti-inflammatory properties, and demonstrated that each 1-point increment in the MD score was associated with a 10% reduction in the risk of CKD [15, 61].

While our analysis found a positive link between DII and low-eGFR risk, studies investigating the relationship between DII and creatinine, uric acid, and BUN yielded inconsistent results. This may be due to the close relationship between eGFR and kidney function, especially in the early stages of CKD. In contrast, other indicators of kidney function are more likely to be influenced by factors such as diet, sex, ethnicity, and muscle mass [62]. Additionally, some indicators of kidney function tend to show changes only at advanced stages of CKD such as creatinine which is influenced when renal function decreases by 50% [62].

Although earlier studies have suggested a link between DII, inflammation and CKD, the exact mechanisms remain unclear. Proposed mechanisms for the connection between DII, inflammation and CKD are mainly focus on high energy, fat, sugar, and protein intake. High-calorie and high-fat diets are known contributors to obesity, which in turn can initiate chronic low-grade inflammation, marked by elevated serum C-reactive protein (CRP) levels [63]. Additionally, adipose tissue secretes various lipid mediators and cytokines, including tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which have been linked to CKD [26, 64–66]. These inflammatory factors are closely linked to the nuclear factor-κB (NF-κB), and TGF-β/Smad3 signaling pathways [67–69]. These pathways lead to increased expression of pro-inflammatory and fibrotic genes, promoting renal inflammation, accumulation of extracellular matrix, and fibrosis [67, 70, 71]. Over time, the persistent activation of these pathways contributes to glomerulosclerosis, tubular atrophy, and progressive kidney dysfunction, ultimately driving the development and progression of chronic kidney disease. Furthermore, a prolonged high-sugar diet leads to hyperglycemia, which produces advanced glycation end-products (AGEs). Diets rich in protein, particularly those high in meat cooked at high temperatures, also contain high amounts of AGEs [72]. These AGEs trigger inflammation, promote insulin resistance, and induce kidney damage at both the glomerular and tubular levels [73–75].

However, it is important to note that dietary patterns alone are unlikely to cause kidney damage in most individuals. It is possible that the association between diet and the onset of CKD may be primarily mediated by insulin resistance and development of the metabolic syndrome, diabetes, and hypertension [76–78]. In fact, these conditions are known to create an environment of chronic inflammation, oxidative stress, and impaired vascular function, which collectively contribute to kidney damage [79–82]. Insulin resistance, for example, can lead to hyperglycemia and increased AGE formation, while hypertension directly strains the renal blood vessels, promoting glomerular injury [82, 83]. Thus, the interplay between diet, metabolic disorders, and kidney health highlights the importance of managing metabolic risk factors in the prevention of CKD.

The present study has several strengths. To our knowledge, this is the first comprehensive systematic review and meta-analysis on the relationship between DII and kidney function. In comparison to previous meta-analysis on the association between DII and CKD, this study included more relevant studies [58]. We conducted a comprehensive search strategy, allowing us to assess various kidney function indicators. To consider the effect of various confounders and potential sources of heterogeneity, only the fully adjusted models were enrolled in the analysis. However, this study has some limitations which should be taken into consideration when interpreting the results. Firstly, dietary intakes were collected by self-reported questionaries which are prone to recall bias. Furthermore, there is a possibility of measurement errors and misclassification of participants in the results due to variations in dietary intake questionnaires which consequently can affect the results. Secondly, while the analysis utilized the most adjusted estimates available, it is important to note that due to the observational nature of the included studies, the possibility of residual and unknown confounders influencing the results cannot be entirely eliminated. Furthermore, a notable limitation of this meta-analysis is the presence of significant publication bias. However, even after conducting the trim and fill analysis, the results did not change considerably. Finally, since the findings are primarily based on cross-sectional studies, establishing a causal relationship between the DII and the outcomes is not possible. Moreover, as only one cohort study was included in the meta-analysis, we were unable to perform a subgroup analysis based on study design. This highlights the need for further prospective cohort studies to better assess these relationships.

In conclusion, this meta-analysis demonstrated a significant positive association between DII and the odds of CKD and low-eGFR. Furthermore, the majority of studies suggest a negative correlation between DII and eGFR. However, the findings regarding the correlation between DII and serum biomarkers of kidney function are inconclusive. Large-scale prospective cohort studies are required to confirm these findings, particularly by assessing different indicators of kidney function.

Abbreviations

DII	Dietary inflammatory index
CKD	Chronic kidney disease
GFR	Glomerular filtration rate
low-eGFR	Low estimated glomerular filtration rate
PRISMA	Preferred reporting items for systematic reviews and meta-analyses
OR	Odds ratio
CL	Confidence intervals
BUN	Blood urea nitrogen (BUN)
RR	Relative risk
HR	Hazard ratio
FFQ	Food frequency questionnaires
MD	Mediterranean diet
CRP	C-reactive protein
TNF-α	Tumor necrosis factor-alpha
IL-6	Interleukin-6
NF-κB	Nuclear factor-κB

Supplementary Information

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Supplementary Material 1

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Author contributions
A.A.K. and F.H. conceived and designed the study. F.H. conducted the statistical analyses. A.A.K. and F.K. interpreted the results and drafted the manuscript. N.S.A. and F.H. provided scientific and language editing of the manuscript. All authors reviewed and approved the final manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Competing interests
The authors declare no competing interests.

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