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# The Association between the Dietary Inflammatory Index and Breast Cancer Risk: An Updated Systematic Review and Meta-Analysis

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**ADMINISTRATIVE INFORMATION**

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**Conflicts of interest -** None declared.

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**Amendments -** This protocol was registered with the International Platform of Registered Systematic Review and Meta-Analysis Protocols (INPLASY) on 19 January 2026 and was last updated on 19 January 2026.

## INTRODUCTION

**Review question / Objective** This meta-analysis aimed to clarify the relationship between the Dietary Inflammatory Index (DII) and the risk of breast cancer (BC) , thereby providing an evidence-based foundation for the primary prevention and secondary management of BC.

**Condition being studied** In recent years, chronic inflammation has been recognized as a core mechanism in cancer development and progression . As a modifiable factor, diet can promote or suppress systemic inflammatory status through its nutritional components, thereby influencing cancer risk. The Dietary Inflammatory Index (DII) serves as a quantitative measure to evaluate the capacity of an individual's dietary intake to modulate inflammatory processes. In recent years, this instrument has been extensively utilized in epidemiological research to elucidate the connections between nutritional habits and both the development and clinical course of breast cancer.

The development of the DII relies on empirical data regarding how specific dietary constituents influence established inflammatory biomarkers. This methodology offers a standardized approach to quantify whether an individual's overall diet tends to promote or suppress systemic inflammation. Supporting this framework, a systematic review by Chen et al. demonstrated a positive correlation between elevated DII scores and increased breast cancer susceptibility, with this relationship being especially marked in premenopausal populations. Corroborating these findings, an extensive Italian case-control investigation revealed that women whose dietary patterns placed them in the highest DII quintile exhibited a substantially greater risk of developing breast cancer compared to those in the lowest quintile. Similarly, positive associations between DII scores and BC risk, especially for specific molecular subtypes such as hormone receptor-positive tumors, have been observed in female

populations in China , Iran , and Korea . Prospective cohort studies, such as the French E3N cohort and the Swedish Women's Lifestyle and Health study , also support the conclusion that a pro-inflammatory diet increases BC risk in postmenopausal women. Wang et al., in the PLCO cohort study, found that maintaining an anti-inflammatory diet after diagnosis was associated with reduced BC-specific mortality. Furthermore, Jang et al. observed among Korean BC patients that higher post-operative DII scores were associated with increased risks of cancer recurrence and overall mortality, especially in younger, obese, and node-positive patients .

Although numerous studies have investigated the relationship between DII and BC risk/prognosis, the findings are not entirely consistent, and there is a lack of systematic integration of the most recent evidence. Some studies reported no significant association, or observed correlations only in specific subpopulations. Furthermore, substantial heterogeneity exists across studies regarding design, population characteristics, dietary assessment methods, and statistical models, limiting comparability and the reliability of conclusions. Consequently, the conduct of an updated systematic review and meta-analysis is necessitated. This endeavor aims to provide a comprehensive synthesis of the evidence linking the DII to breast cancer risk and prognostic outcomes, while also investigating the origins of observed heterogeneity across studies.

This study aims to systematically review and meta-analyze relevant literature published up to 2025 to assess the relationship between the Dietary Inflammatory Index and the risk of BC development and its prognosis, thereby providing evidence-based support for the primary prevention and secondary management of BC.

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## METHODS

**Participant or population** Female individuals, regardless of race or geographical region, with a pathologically confirmed diagnosis of breast cancer.

**Intervention** This meta-analysis synthesizes evidence from observational studies; therefore, no active intervention was administered. The primary exposure of interest is the Dietary Inflammatory Index (DII) or its energy-adjusted version (E-DII).

**Comparator** The comparator group is defined as participants with lower DII/E-DII scores, indicative of an anti-inflammatory dietary pattern. This group corresponds to those in the lowest quantile (e.g., quartile 1 or quintile 1) of DII distribution in the included studies.

**Study designs to be included** Observational studies (cohort, case-control, cross-sectional); studies must provide extractable effect estimates (Odds Ratio [OR], Relative Risk [RR], Hazard Ratio [HR]) with corresponding 95% Confidence Intervals (CIs).

**Eligibility criteria** The inclusion criteria were as follows:

(1) Study type: Observational studies (cohort, case-control, cross-sectional); studies must provide extractable effect estimates (Odds Ratio [OR], Relative Risk [RR], Hazard Ratio [HR]) with corresponding 95% Confidence Intervals (CIs).

(2) Participants: Female individuals, regardless of race or geographical region, with a pathologically confirmed diagnosis of breast cancer.

(3) Exposure: DII or its energy-adjusted version (E-DII). The DII/E-DII must be reported either as a continuous variable or as a categorical variable (e.g., quantiles).

(4) Outcome measures: BC risk (HR) during follow-up. Studies reporting only on prevalence or prognostic outcomes (e.g., survival, recurrence) without incidence data were excluded from the quantitative meta-analysis.

Exclusion criteria included: animal or in vitro studies, commentaries, conference abstracts, studies not providing original data or where data could not be obtained after contacting the authors. For duplicate publications, the version with the most comprehensive data or the most recent publication was retained.

**Information sources** PubMed, Embase, Web of Science, and the CochraneLibrary.

**Main outcome(s)** Pooled Odds Ratio (OR) with 95% confidence interval (CI) for case-control studies, comparing the highest versus lowest category of Dietary Inflammatory Index (DII/E-DII).

Pooled Hazard Ratio (HR) with 95% confidence interval (CI) for cohort studies, comparing the highest versus lowest category of DII/E-DII.

Dose-response relationship, expressed as the pooled risk estimate (OR/HR) associated with a one-unit increase in the continuous DII score.

**Quality assessment / Risk of bias analysis** Two reviewers independently assessed the methodological quality of the included cohort and case-control studies using the Newcastle-Ottawa Scale (NOS), as recommended by the Agency for Healthcare Research and Quality (AHRQ). The NOS evaluates studies across three domains: (1) selection of study groups, (2) comparability of groups, and (3) ascertainment of either the exposure (for case-control studies) or outcome (for cohort studies). A star system is used to award points within each domain, with a maximum possible score of 9 stars, indicating the highest methodological quality. Disagreements in quality assessment were resolved by consensus.

**Strategy of data synthesis** (1) Effect Size Pooling: For dichotomous outcomes (e.g., disease risk, mortality risk), fully adjusted ORs or HRs with their 95% CIs were extracted and pooled. Effect sizes comparing different DII/E-DII quantiles (e.g., Q4 vs. Q1) were considered the primary analysis. If studies only provided effect estimates for DII as a continuous variable, the OR/HR corresponding to a one-unit increase in the DII score was pooled. A random-effects model (DerSimonian and Laird method) was used to pool effect sizes, accounting for potential heterogeneity among studies.
(2) Assessment of Heterogeneity: The degree of variability among the included studies was quantified using Cochrane's Q test (with a significance threshold of $\alpha$=0.10) and the $I^2$ statistic. The magnitude of heterogeneity was categorized as follows: $I^2$ values of 25%, 50%, and 75% represented low, moderate, and high heterogeneity, respectively. A random-effects model was implemented for all pooled analyses to account for expected methodological and population variations.
(3) Subgroup and Sensitivity Analyses: Pre-specified subgroup analyses were conducted to investigate potential effect modifiers, including study design (cohort versus case-control) and menopausal status (premenopausal versus postmenopausal). Additional sensitivity analyses were performed to evaluate the robustness of the primary findings.
(4) Evaluation of Publication Bias: Potential publication bias was examined through visual inspection of funnel plots for analyses including at least 10 studies, as the statistical power of such tests is limited with a smaller number of studies. For analyses with fewer studies, publication bias was not formally assessed due to low test power. When presented, asymmetry in the funnel plot was interpreted as suggestive of possible publication bias, acknowledging that other factors (e.g., heterogeneity, methodological differences) could also cause asymmetry.Potential publication bias was examined through visual inspection of funnel plots. Asymmetrical distribution of effect estimates in the funnel plot was interpreted as indicative of possible publication bias.
(5) Dose-Response Analysis: For studies that reported the DII as a continuous variable, the adjusted OR or HR for a one-unit increase in the DII score was directly extracted and pooled. These estimates were derived from regression models (logistic regression for case-control studies, Cox proportional hazards regression for cohort studies) that treated DII as a continuous linear predictor, adjusting for relevant confounders. For studies that reported DII in categories (e.g., quartiles) and did not provide a per-unit estimate, the continuous effect size could not be derived. The pooled effect per one-unit DII increment was therefore calculated by synthesizing the estimates from studies that provided continuous data, using the random-effects model described above.
All statistical computations were executed using R software (version 4.2.0) with the "metafor" package. Statistical significance was defined as a two-tailed P-value of less than 0.05.

**Subgroup analysis** Pre-specified subgroup analyses were conducted based on:

Study Design: Comparing results from case-control studies versus prospective cohort studies.

Menopausal Status: Stratifying the analysis of case-control studies by participant menopausal status into:

Premenopausal women

Postmenopausal women

Women of unspecified menopausal status (mixed population)

These subgroup analyses were performed to explore potential sources of heterogeneity and to assess whether the association between DII/E-DII and breast cancer risk differed across these key population and methodological characteristics.

**Sensitivity analysis** Sensitivity analyses were performed to evaluate the robustness of the primary findings. These analyses assessed the

influence of methodological variations in the included studies, particularly regarding:

Method of DII Categorization: The impact of different DII grouping strategies (e.g., dichotomization, tertiles, quartiles, quintiles) on the pooled effect estimates was examined within the subgroup analyses by menopausal status.

Statistical Model: The consistency of results was confirmed through the use of a random-effects model for all pooled analyses to account for expected heterogeneity.

Study Quality: Although not explicitly detailed as a sensitivity analysis using a quality threshold, the methodological quality of all included studies was assessed using the Newcastle-Ottawa Scale (NOS), and the analysis included studies of moderate to high quality (NOS score ≥ 6).

**Country(ies) involved** China.

**Keywords** Dietary Inflammatory Index, Breast cancer, Meta-analysis, Nutritional intervention.

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