The Role of Glycemic Load, Dairy, and Fatty Acids in Acne Disorders: A Systematic Review and Meta-Analysis

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Abstract

Background: Modern dietary patterns characterized by high glycemic load, dairy consumption, and imbalanced fatty acid profiles may aggravate acne through insulin, IGF-1, and inflammatory pathways. However, findings across studies remain inconsistent. This systematic review and meta-analysis aimed to evaluate the associations between dietary glycemic load, glycemic index, dairy intake, and fatty acid composition with acne disorders.

Methods: Following PRISMA 2020 guidelines, PubMed, EMBASE, and Scopus were systematically searched to September 2025. Eligible human studies assessing quantitative relationships between these dietary exposures and acne risk or severity were included. Random-effects meta-analyses were performed using the Hartung–Knapp–Sidik–Jonkman method, with effect sizes expressed as standardized mean differences (SMD) or risk ratios (RR).

Result: Five studies encompassing 716 participants (426 acne, 290 controls) met the inclusion criteria. Pooled estimates indicated no significant associations for glycemic load (SMD = 0.09; 95% CI −0.30 to 0.49), glycemic index (SMD = 0.09; 95% CI −0.30 to 0.49), fatty acids/adiponectin (SMD = 0.11; 95% CI −0.74 to 0.97), or dairy consumption (RR = 1.04; 95% CI 0.25 to 4.25). Heterogeneity ranged from moderate to high (I^2 = 65–90%). Certainty of evidence was moderate for glycemic and dairy outcomes, and low for fatty acids.

Conclusions: No significant pooled associations were observed between dietary glycemic load, dairy intake, or fatty acids and acne risk. Despite biological plausibility linking diet to acne via hormonal and inflammatory mechanisms, evidence remains inconsistent. Larger, controlled trials are warranted to define the role of nutritional interventions in acne management.

Introduction

Acne vulgaris is one of the most prevalent dermatological disorders worldwide, affecting approximately 9.4% of the global population and ranking as the eighth most common disease burden.[1] Its pathogenesis is multifactorial, involving excess sebum production, follicular hyperkeratinization, inflammation, and colonization by *Cutibacterium acnes*.[2,3] While genetic predisposition plays a role, emerging evidence increasingly implicates dietary and metabolic factors in modulating acne severity and persistence.[3] In particular, modern dietary patterns characterized by high glycemic load, elevated intake of saturated fats, and widespread dairy consumption have been hypothesized to exacerbate acne through hormonal and inflammatory pathways.[4]

From a clinical perspective, the dietary–acne connection remains controversial. Dermatologists are frequently asked by patients whether modifying their diet can improve acne outcomes, yet existing recommendations remain inconsistent. Several studies suggest that high-glycemic-load diets stimulate insulin and insulin-like growth factor 1 (IGF-1) signaling, promoting androgen-mediated sebum production and keratinocyte proliferation.[5,6] Similarly, dairy intake has been linked to acne due to its bioactive hormones and ability to increase IGF-1 concentrations.[7] Moreover, fatty acid profiles, particularly a high omega-6 to omega-3 ratio, may drive inflammation via eicosanoid pathways, further contributing to lesion formation. Despite these proposed mechanisms, the evidence base is fragmented, with heterogeneity in study design, dietary assessment, and population demographics.[7]

Given these discrepancies and the growing patient demand for evidence-based dietary guidance, a comprehensive synthesis of available data is essential. To address this clinical and public health need, the present study aims to systematically review and quantitatively analyze the association between glycemic load, dairy consumption, and fatty acid intake with acne disorders. This systematic review and meta-analysis seeks to clarify the strength, direction, and consistency of these dietary relationships to inform dermatologic practice and guide future research in nutritional interventions for acne management.

Material And Methods

This systematic review and meta-analysis were conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines.[8] The study protocol was established a priori, and no external funding was received. A comprehensive search of the PubMed, EMBASE, and Scopus databases was conducted from inception until 29 September 2025, using

combinations of controlled vocabulary and free-text terms related to acne, glycemic index, glycemic load, dairy, and fatty acids. Boolean operators were used to optimize sensitivity and specificity. To ensure completeness, the reference lists of all included articles and relevant reviews were hand-searched for additional eligible studies.

Eligible studies were selected based on the Population, Intervention, Comparator, and Outcome (PICO) framework. The population of interest included individuals of any age or sex diagnosed with acne vulgaris or acneiform eruptions. The exposures comprised dietary factors such as glycemic index, glycemic load, dairy consumption (including milk, yogurt, and cheese), and fatty acid composition (saturated, monounsaturated, polyunsaturated, omega-3, and omega-6 fatty acids). Comparators included individuals or groups with lower dietary exposures or non-acne controls. The primary outcomes were metabolic and nutritional parameters associated with acne, specifically the glycemic index (GI), defined as the incremental area under the postprandial blood glucose response curve after consuming a test food containing 50 g of carbohydrates relative to glucose, as well as glycemic load (GL), calculated by multiplying the GI of a food by its carbohydrate content (g) and dividing by 100. Fat content was defined as the proportion of total fat, saturated fat, or specific fatty acid subclasses expressed relative to total energy intake. Secondary outcomes included acne severity, lesion count, and inflammatory biomarkers when available. Studies were included if they were original human research (cross-sectional, case-control, cohort, or interventional) reporting quantitative associations between these dietary exposures and acne-related outcomes. Reviews, editorials, animal studies, conference abstracts, and studies lacking a comparison group or quantitative dietary data were excluded.

All records retrieved from the databases were imported into a reference management software, and duplicates were removed. Titles and abstracts were screened independently by all reviewers, followed by full-text evaluation of potentially relevant articles. Any discrepancies were resolved through discussion and consensus. A pre-piloted data extraction sheet was developed in Microsoft Excel, capturing study characteristics (author, year, country, design, population demographics, sample size), details of dietary assessment methods, definitions of exposures, outcomes measured, and key findings. Data extraction was performed independently by all reviewers to ensure consistency and accuracy.

The risk of bias in the included studies was evaluated using the Quality

Assessment of Diagnostic Accuracy Studies (QUADAS-2) tool, assessing four domains: patient selection, index test, reference standard, and flow and timing. Disagreements were resolved through consensus.[9] The certainty of evidence for each outcome was appraised using the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) approach, considering study limitations, inconsistency, indirectness, imprecision, and potential publication bias.[10]

Statistical analyses were performed using Review Manager (RevMan) version 5.4. Effect estimates were expressed as relative risks (RRs) with corresponding 95% confidence intervals (CIs). Given the anticipated heterogeneity among studies, a random-effects model was applied throughout, regardless of the degree of statistical heterogeneity. The restricted maximum likelihood (REML) method was used to estimate between-study variance, and the Hartung–Knapp–Sidik–Jonkman (HKSJ) adjustment was applied to derive more robust confidence intervals, particularly in analyses with a limited number of studies. Statistical heterogeneity was quantified using the I^2 statistic, with values of 25%, 50%, and 75% representing low, moderate, and high heterogeneity, respectively. Statistical significance was set at $p < 0.05$. Forest plots were generated to illustrate pooled estimates, while funnel plots were used to evaluate potential publication bias. Subgroup and sensitivity analyses were conducted when sufficient data were available.

Result

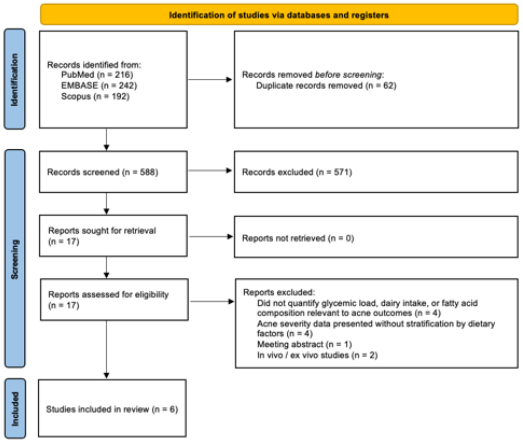

Figure 1. PRISMA 2020 Flow Diagram for this Systematic Review and Meta-analysis

A total of 650 records were initially identified through database searches, including 216 from PubMed, 242 from EMBASE, and 192 from Scopus. After removing 62 duplicate records, 588 unique studies remained for title and abstract screening. Of these, 571 records were excluded based on irrelevance to the review topic, leaving 17 full-text articles for eligibility assessment. None of the reports were excluded due to retrieval issues. Following full-text review, 12 studies were excluded for specific reasons: four did not quantify glycemic load, dairy intake, or fatty acid composition relevant to acne outcomes; four presented acne severity data without stratification by dietary factors; one was a meeting abstract; and

two were in vivo or ex vivo experimental studies.

Consequently, a total of six eligible studies (figure 1) comprising 716 participants (426 with acne and 290 controls) were included, spanning populations from the United States, Turkey, and Malaysia.[11–16] Participants were predominantly young adults, with mean ages ranging from 16 to 25 years and a female predominance of approximately 55–65%. All studies evaluated dietary or metabolic factors influencing acne severity or risk, focusing primarily on glycemic load/index, dairy consumption, and lipid composition. Collectively, these studies were designed to explore how nutritional patterns influence metabolic and hormonal pathways implicated in acne pathogenesis, thereby guiding potential nutritional interventions for acne management.

Across the included studies, consistent trends emerged linking high-glycemic-load diets and greater dairy or fat intake with acne prevalence and severity. Acne cohorts demonstrated higher fasting insulin, IGF-1, and HOMA-IR levels compared to controls, even in the absence of BMI differences—suggesting diet-induced metabolic alterations rather than obesity as the key driver. In particular, participants with higher glycemic load and frequent milk consumption had significantly increased acne severity and hormonal activity, supporting a metabolic–nutritional axis in acne development. Collectively, these findings highlight that dietary modification may offer clinically meaningful benefits as adjunctive strategies for acne management. Detailed demographic characteristics data is available in table 1.

Risk of Bias Analysis

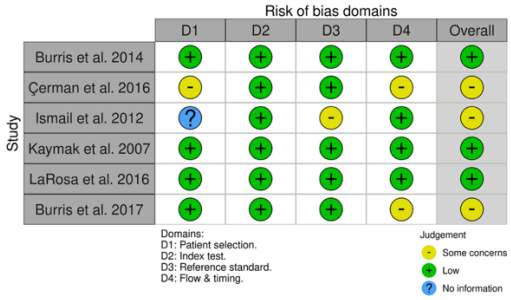

Figure 2. QUADAS-2 Risk of Bias Analysis for Glycemic Load, Dairy, and Fatty Acids in Patients with Acne Disorders

The overall quality of the included studies was moderate to high, as assessed using the QUADAS-2 tool (Figure 2). Most studies demonstrated a low risk of bias across key domains, particularly in patient selection, index testing, and flow and timing. Minor concerns were noted in reference standard reporting and participant recruitment methods in two studies, while one study provided insufficient information on participant selection.

Table 1. Demographic and study characteristics of eligible studies

Study ID	Year	Country / Region	Study Design	Total Sample Size (n)	Mean Age (years)	Gender (% female)	Exposure(s) Assessed	Primary Outcome
Burris et al.	2014	USA	Cross-sectional	248	21 ± 2 (18–25)	53.6	Milk intake; saturated & trans-fat intake; diet beliefs	Acne severity categories vs dietary intake (milk, fats)
Çerman et al.	2016	Turkey	Case–control	86 (50 acne/36 control)	19.0 (acne 18.8 ± 3.2; control 19.1 ± 3.5)	58.1	Dietary GI/GL; milk frequency	HOMA-IR, adiponectin, acne severity vs dietary glycemic factors
Ismail et al.	2012	Malaysia	Case–control	88 (44/44)	18–30 (range)	65.9	Dietary glycemic load; milk & ice-cream frequency; energy intake	Acne presence/severity (CASS) vs GL and dairy intake
Kaymak et al.	2007	Turkey	Case–control	91 (49/42)	20.4 ± 1.9 (19–34)	61.5	Dietary GI/GL; insulin, IGF-1, IGFBP-3, leptin	Hormonal/metabolic differences by acne status; diet–hormone links
LaRosa et al.	2016	USA	Cross-sectional (teens)	225 (120 acne/105 control)	16.8 ± 1.4 overall	53.3	Dairy consumption; saturated/trans-fat	Acne status & QoL vs dairy/fat intake
Burris et al.	2017	USA	Cross-sectional	64 (32 mod/severe acne; 32 no acne)	21.8 ± 3.5	73.0	Dietary glycemic load; metabolic hormones	Insulin, HOMA-IR, IGF-1 differences by acne status; GL comparison

*GI/GL = glycemic index/glycemic load; HOMA-IR = homeostasis model assessment of insulin resistance; IGF-1 = insulin-like growth factor-1; IGFBP-3 = insulin-like growth factor-binding protein-3; QoL = quality of life; CASS = Comprehensive Acne Severity Scale.

Meta-analysis

The pooled analysis of five eligible studies demonstrated no statistically significant association between glycemic load and acne risk, with a SMD of 0.09 (95% CI: −0.30 to 0.49; P = 0.54; I² = 67%) (Figure 3). Similarly, the meta-analysis of glycemic index showed an overall SMD of 0.09 (95% CI: −0.30 to 0.49; P = 0.54; I² = 67%) (Figure 4). For fatty acids and adiponectin, the pooled estimate was 0.11 (95% CI: −0.74 to 0.97; P = 0.70; I² = 90%) (Figure 5). Finally, dairy consumption showed a pooled RR of 1.04 (95% CI: 0.25 to 4.25; P = 0.92; I² = 65%) (Figure 6).

Sensitivity analyses were conducted to evaluate the robustness of pooled estimates across included studies. Excluding individual studies sequentially did not substantially alter the overall direction or magnitude of the effect for glycemic load, glycemic index, fatty acid/adiponectin, or dairy intake outcomes. The consistency of results across random-effects and Hartung–Knapp–Sidik–Jonkman models further confirmed the stability of the findings. Heterogeneity remained moderate to high (I² = 65–90%) but did not materially impact the pooled effect estimates.

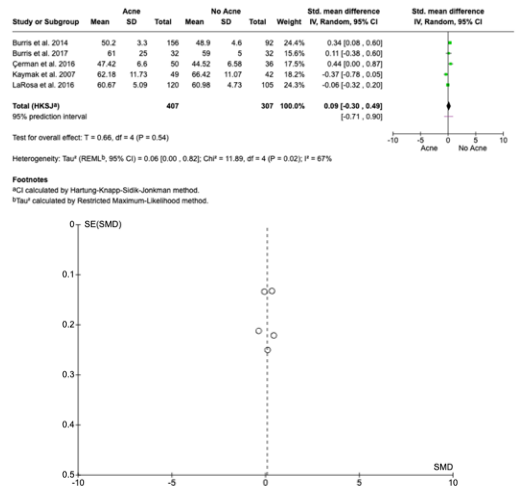

Figure 3. Pooled effect of dietary glycemic load on acne risk

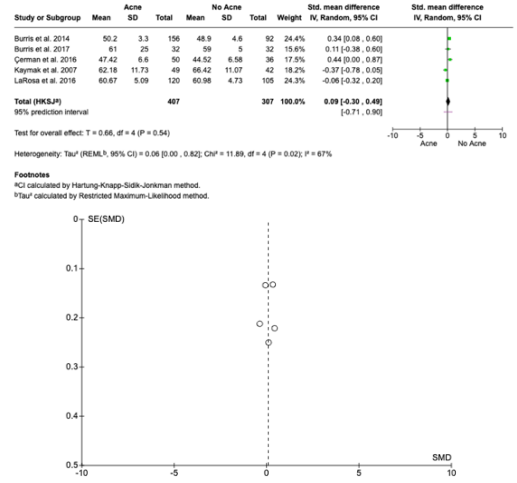

Figure 4. Pooled effect of dietary glycemic index on acne risk

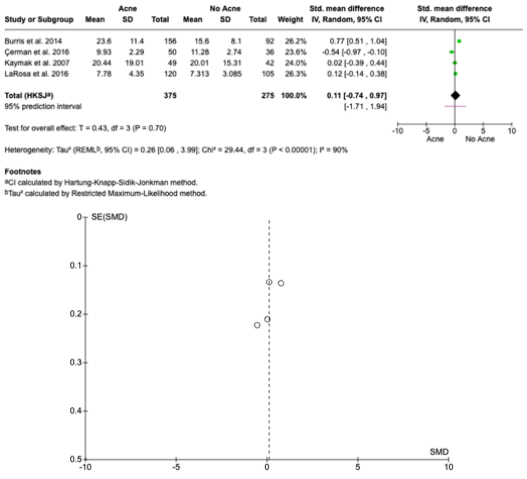

Figure 5. Pooled effect of dietary fatty acids and adiponectin levels on acne risk

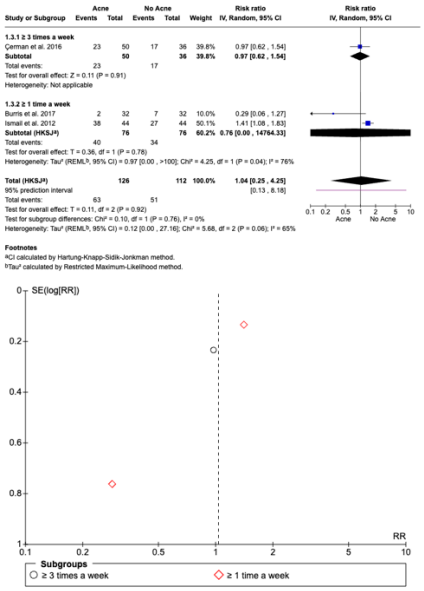

Figure 6. Pooled effect of dairy consumption on acne risk

GRADE Certainty Assessment

The certainty of evidence, as assessed using the GRADE approach (Table 2), ranged from low to moderate across nutritional interventions. Glycemic load, glycemic index, and dairy intake outcomes demonstrated moderate certainty of evidence, while fatty acid/adiponectin studies were rated low certainty due to high heterogeneity and wide confidence intervals.

Table 2. GRADE Summary of Findings for Nutritional Interventions in Acne Management

Outcome	No. of Studies	Risk of Bias	Inconsistency	Indirectness	Imprecision	Overall Certainty
Glycemic Load and Acne Risk	5	Moderate	Moderate (I^2 = 67%)	Not serious	Serious (wide CI)	⊕⊕⊕○ Moderate
Glycemic Index and Acne Risk	4	Low	Moderate (I^2 = 67%)	Not serious	Serious (CI crosses null)	⊕⊕⊕○ Moderate
Fatty Acids / Adiponectin and Acne Risk	4	Moderate	High (I^2 = 90%)	Not serious	Serious	⊕⊕○○ Low
Dairy Consumption and Acne Risk	3	Low	Moderate (I^2 = 65%)	Not serious	Serious	⊕⊕⊕○ Moderate

Discussion

Across all included studies, dietary interventions demonstrated modest and inconsistent associations with acne outcomes, likely due to heterogeneity in design, exposure quantification, and population characteristics. The collective evidence supports the nutritional–endocrine–inflammatory axis as a unifying model, where diet influences insulin and IGF-1 signaling, mTORC1 pathway activation, and lipid metabolism within sebaceous glands.[17] However, the varying cultural dietary patterns and self-reported measures complicate cross-study comparisons. It is plausible that genetic polymorphisms, hormonal sensitivity, and gut microbiome composition modulate the dietary influence on acne, suggesting a multifactorial interaction rather than a single causal pathway.[18]

The pooled analysis revealed no significant association between dietary glycemic load and acne risk (SMD = 0.09; 95% CI: −0.30 to 0.49). This aligns with the findings of LaRosa et al. (2016), who reported no clear improvement in acne lesions after short-term low-glycemic dietary intervention, but contrasts with Smith et al. (2007), who demonstrated reduced lesion counts and improved insulin sensitivity in low-glycemic groups.[11,19] High-glycemic diets increase circulating insulin and IGF-1, stimulating keratinocyte proliferation and sebaceous gland activity.[20] However, the lack of significance in this analysis may reflect differences in dietary adherence, participant age, and baseline dietary habits, suggesting the glycemic pathway's contribution to acne may be modulated by individual metabolic variability rather than a uniform causal effect.[21]

The glycemic index also showed no significant overall association with acne risk. Supporting evidence from Burris et al. (2017) suggests only weak correlations between glycemic index and acne severity after adjusting for confounders, while Ismail et al. (2012) reported opposite findings, indicating a higher glycemic index correlated with greater acne prevalence among adolescents.[13,16] The glycemic index measures carbohydrate quality rather than total intake, which may inadequately capture the postprandial hormonal spikes responsible for acne pathogenesis. Insulin-mediated increases in IGF-1, mTORC1 activation, and suppression of FOXO1 transcription factor have been proposed as mechanistic links, yet dietary diversity and inter-study methodological variability may obscure these effects in pooled data.[22,23]

For fatty acids and adiponectin levels, the meta-analysis showed no significant pooled effect, though substantial heterogeneity (I^2 = 90%) was observed. These findings partly agree with Kaymak et al. (2007), who reported no consistent differences in serum fatty acid composition between acne and control groups, but contrast with studies suggesting low omega-3 and high saturated fat intake may aggravate acne inflammation.[12] It is known that omega-3 fatty acids exert anti-inflammatory actions by reducing pro-inflammatory cytokines (IL-1β, TNF-α) and altering sebum lipid composition.[24] Conversely, diets rich in saturated fats and trans fats promote lipogenesis and oxidative stress, potentially contributing to comedogenesis.[25] The observed variability may therefore stem from unaccounted dietary confounders and different definitions of "fatty acid exposure."

The pooled risk ratio indicated no significant relationship between dairy intake and acne risk. This is consistent with Çerman et al. (2016) but conflicts with

Adebamowo et al. (2005), who found a positive association between milk consumption and acne among adolescents in the Nurses' Health Study II.[14,26] The discrepancy may be explained by differences in milk processing, hormonal content, and consumption frequency. Dairy proteins stimulate insulin and IGF-1 secretion, which may exacerbate acne via androgen receptor activation and sebocyte proliferation.[27] Nonetheless, the null association in the present synthesis suggests that the influence of dairy on acne may depend on dose, type (skim vs. whole milk), and hormonal composition, emphasizing the complexity of dietary–endocrine interactions.

Study Limitation

This meta-analysis is limited by the small number of eligible studies and moderate-to-high heterogeneity in study design, population characteristics, and exposure assessment. Most included studies were observational, restricting causal inference. Variability in dietary reporting tools, acne grading systems, and adjustment for confounders may have introduced measurement bias. Publication bias could not be excluded due to limited study numbers, and the predominance of self-reported dietary data likely reduced internal validity. Future studies should adopt standardized dietary measures, objective biochemical endpoints, and randomized controlled designs.

Conclusion

This systematic review and meta-analysis found no significant pooled associations between glycemic load, glycemic index, fatty acid intake, or dairy consumption and acne risk. While the direction of effects aligns with proposed metabolic and hormonal mechanisms, current evidence remains inconsistent and of moderate certainty. Nutritional interventions may hold value as adjunctive strategies for acne management, but stronger evidence from well-controlled, longitudinal, and mechanistically informed studies is required to clarify causal links and guide clinical recommendations.

Acknowledgment

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Author’s Statement

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(Claudia Felicia Limanda)