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DIETARY INFLUENCES ON ACNE VULGARIS: INSIGHTS FROM KEY NUTRITIONAL MODELS

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ABSTRACT

Introduction: Acne vulgaris (AV) is a prevalent inflammatory dermatosis with substantial psychosocial burden. Diet can modulate AV via insulin/IGF-1/mTORC1 signalling, inflammation, lipid metabolism, and oxidative stress, yet many patients self-modify diet without structured guidance.
Aim: To synthesise how key dietary patterns and components influence acne biology and clinical severity, and to distil pragmatic counselling points.
Materials and Methods: Structured analysis of PubMed (2020–2025) using "acne" AND "diet." A broad range of study types was considered. Priority was given to evidence on AV in populations without comorbid conditions (e.g., PCOS). The synthesis focused on dietary patterns and specific food groups; studies centred primarily on vitamins, supplements, or the microbiome were not the focus.
Results: Glycaemic load (GL) is a key modifiable driver. Low-GL patterns show the strongest clinical signal and are directly actionable. Higher intake of sugary drinks, sweet snacks, and refined grains aligns with greater acne burden, consistent with insulin–mTORC1 activation. Mediterranean-style eating associates with lower severity and favourable metabolic markers. Plant-forward diets appear beneficial when emphasising whole-food, low-GL choices; veganism per se and routine gluten-free eating lack support without specific indications. Dark chocolate may aggravate lesions in some individuals through non-glycaemic mechanisms. Dairy may warrant cautious, personalised reduction while maintaining micronutrient adequacy. Shifting fat quality toward MUFA/omega-3s and away from saturated fat aligns with lower inflammatory burden.
Conclusions: Dietary counselling should complement dermatological therapy. Low-GL and Mediterranean-style patterns show the most consistent associations with reduced acne burden; rigorous trials of whole-diet interventions remain needed.

KEYWORDS

Acne Vulgaris, Dairy Intake, IGF-1/mTORC1 Signalling, Low-Glycaemic-Load Diet, Mediterranean Diet, Nutritional Counselling

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Introduction

Acne vulgaris (AV) is among the most prevalent chronic inflammatory skin conditions, affecting up to 80–100% of individuals during adolescence and often persisting into adulthood [1]. Beyond its visible manifestations, AV has a considerable psychosocial impact, negatively affecting self-perception, interpersonal relationships, and overall quality of life [2].

The pathogenesis of AV is multifactorial, encompassing excess sebum secretion, follicular hyperkeratinisation, proliferation and metabolic activity of Cutibacterium acnes, and dysregulated cutaneous inflammation [3]. Increasing attention has been directed toward endocrine and metabolic mechanisms, particularly insulin and insulin-like growth factor-1 (IGF-1), which activate mammalian target of rapamycin complex 1 (mTORC1) and promote sebocyte lipogenesis and keratinocyte proliferation [4–6].

For many years, the relationship between diet and acne remained controversial due to heterogeneous methodologies, reliance on self-reported dietary data, and confounding lifestyle variables [7]. However, growing evidence now recognises diet as a relevant modifiable determinant influencing the onset and clinical severity of acne [1].

High-glycaemic-load diets elevate postprandial insulin, enhance IGF-1 signalling, and intensify inflammatory cascades within the pilosebaceous unit [1,4,8]. Dairy intake may further contribute to acne pathophysiology by increasing IGF-1 bioavailability and delivering androgenic precursors and bioactive molecules [1].

In addition to single food groups, Western dietary patterns characterised by high consumption of refined sugars, saturated fats, and an unfavourable omega-6 to omega-3 fatty acid ratio are associated with enhanced systemic inflammation and oxidative stress [9,10]. Conversely, health-promoting dietary patterns enriched with antioxidants, polyphenols, fibre, and omega-3 fatty acids, such as the Mediterranean diet and plant-based models, demonstrate anti-inflammatory and antioxidative effects associated with reduced acne severity [11–14].

Emerging research further supports mechanistic links through the gut–skin axis. Altered gut microbiota composition, reduced microbial diversity, and impaired intestinal barrier integrity may facilitate systemic inflammatory responses contributing to acne progression [15,16]. Despite rising clinical interest, patients frequently implement dietary modifications independently due to a lack of evidence-based nutritional guidelines for acne management [17,18].

To date, many studies have investigated either isolated dietary components (such as dairy products, chocolate, and gluten-containing foods) or single nutrient determinants (including glycaemic load and omega-6 to omega-3 ratios) rather than evaluating whole dietary models [1,8–10,18]. Although these approaches provide valuable mechanistic insights, they do not adequately reflect the complexity of real-world eating behaviours or the potential synergistic effects between foods and nutrients. A critical knowledge gap persists regarding the comparative role of dietary patterns and specific diet-related exposures in acne pathogenesis and therapeutic response.

The purpose of this narrative review is to synthesise recent evidence on the influence of major nutritional models, including low-glycaemic-load dietary patterns, the Mediterranean diet, very low-calorie ketogenic diets (VLCKD), and plant-based diets, as well as key dietary components such as dairy, chocolate, and gluten-containing foods, on the biological mechanisms and clinical severity of acne vulgaris. This review aims to inform the development of personalised, diet-inclusive acne management strategies within clinical practice.

Results

Patient Behaviours: Self-Directed Dietary Changes

A national cross-sectional survey in the United States demonstrated that nearly half of individuals with moderate-to-severe acne (47.5%) reported making at least one dietary modification to improve their skin condition, compared with 42.1% of the overall acne population (n=252) [17].

Among participants with moderate-to-severe acne (n=40), the most frequently adopted dietary strategies were:

- reducing oily, greasy, or fried foods (32.5%)
- reducing sugar intake (27.5%)
- limiting dairy products (17.5%)
- increasing vegetable consumption (12.5%)
- increasing fruit intake (7.5%)

Less commonly reported adjustments included reducing chocolate intake (5.0%) and eliminating whey protein (0.0%). Additional dietary interventions such as ketogenic, Mediterranean, vegan, vegetarian, or paleo eating patterns, intermittent fasting, or increased water intake were collectively applied by 7.5% of participants [17].

These patient-driven changes closely reflect food groups scientifically recognised as relevant to acne pathophysiology, including high-glycaemic-load foods, dairy products, and selected trigger foods [1,8,9,10,19]. However, such interventions are largely self-directed and unstandardised, indicating a lack of clinically implemented dietary guidance. The literature notes that patients are often motivated to modify their eating habits due to the perception of symptom improvement, yet structured, evidence-based nutritional recommendations for acne remain insufficiently established in current dermatological practice [17,18]. Literature reviews highlight the importance of including structured dietary guidance within acne management strategies [6,7].

Mediterranean Diet Pattern

The Mediterranean diet (MD) shows **consistent observational associations** with reduced acne severity. MD focuses on vegetables, fruits, legumes, whole grains, nuts, and olive oil, with limited intake of high-glycaemic processed foods and saturated fats [11–14]. Its anti-acne potential is supported mechanistically through improvements in insulin regulation, decreased IGF-1 bioactivity, and increased antioxidant and anti-inflammatory nutrient density [4–6,11–14].

A university case-control study shows significantly lower acne severity in individuals with strong MD adherence [11]. Iranian case-control data confirmed this association across distinct cultural dietary contexts

[12]. Among French women, greater MD adherence corresponded to lower clinical acne severity [13], indicating relevance in adult female acne.

A biomarker-focused investigation showed that MD-adherent acne patients have reduced serum IGF-1, aligning with reduced mTORC1 activation [20]. Improved phase angle in these patients indicates broader metabolic benefits important for cutaneous physiology [21].

Although no RCTs have yet evaluated MD as a primary acne therapy, clinical guidelines identify MD as a safe, sustainable dietary pattern that **may complement** acne treatment [18].

Clinical interpretation:

MD may be incorporated as part of acne management due to its favourable nutrient profile and alignment with mechanisms relevant to acne pathogenesis.

Plant-Based and Vegan Dietary Models

Plant-forward diets emphasise polyphenols, antioxidants, fibre, and beneficial PUFA, contributing to lower oxidative and inflammatory activity involved in acne [22–24].

Multiple cohort studies report improved dietary quality and acne-related quality of life (QoL) among acne patients increasing plant food intake [25,26]. These improvements appear driven not by the exclusion of animal products per se but by enhanced nutrient quality.

Clinical results from a vegetarian-plus-probiotic intervention demonstrated acne improvement, though dietary attribution remained unclear [15].

A Mendelian randomisation study showed no causal relationship between vegetarian diet assignment and acne risk [27], underscoring that food quality, not exclusion rules, drives benefit.

Concerns arise where “plant-based” models include refined grains and sweetened vegan ultra-processed foods, which maintain a high glycaemic load and can worsen acne [1,4–6].

Clinical interpretation:

Whole-food, low-GL plant-based diets may support acne improvement, but veganism alone is not an evidence-based therapy.

Very Low-Calorie Ketogenic Diet (VLCKD)

VLCKD markedly reduces postprandial insulin, lowering IGF-1/mTORC1 pathway activation [4–6]. Reviews propose VLCKD as a potential adjuvant therapy through improved insulin sensitivity and reduced inflammatory signalling [28].

However, no RCTs have evaluated VLCKD as a direct acne treatment. Clinical nutrition guidelines stress the need for medical oversight, monitoring micronutrient adequacy, and avoiding long-term restriction without justification [18,28].

Clinical interpretation:

VLCKD may benefit patients with insulin dysregulation, but its adoption for acne should remain experimental and supervised.

Glycaemic Load and Specific Triggers (including Chocolate)

Dietary glycaemic load is a central, modifiable driver in acne. An LGL-focused approach shows the most persuasive clinical signal, with randomised evidence for reducing inflammatory lesions following targeted counselling [8]. Consistently, higher consumption of sugary beverages, sweet snacks, and refined grains correlates with greater acne severity in observational datasets [9,10,29]. Mechanistically, high-GL exposures promote insulin surges that potentiate sebaceous lipogenesis, enhance androgen signalling, and amplify perifollicular inflammation through insulin–mTORC1 pathways [4–6]. Quality-of-life studies further link sweet, fried, and sugar-sweetened items with a higher patient-reported burden [25,26]. Notably, dark chocolate (85% cocoa) has been observed to aggravate lesions despite a low GL, suggesting additional non-glycaemic mechanisms may contribute [30].

Clinical interpretation:

Prioritise an **LGL-oriented pattern** by replacing refined carbohydrates and sugar-sweetened drinks with minimally processed, fibre-rich foods; pair carbohydrates with protein/fat/fibre to blunt glycaemic excursions. Use an **individualised, time-limited trial** for potential triggers (including dark chocolate), monitor flare patterns, and adjust accordingly.

Dairy Intake and Practical Triggers

Dairy intake, particularly skimmed milk and sweetened milk beverages, consistently correlates with higher acne prevalence and severity across populations [9,10]. Proposed mechanisms include androgen precursors, enhanced IGF-1 release, and follicular hyperkeratinisation [1,31].

Among adult females, dairy intake showed a stronger association with acne severity than in mixed-gender cohorts, suggesting heightened hormonal responsiveness [31].

Dairy products high in added sugars may worsen acne through dual hormonal and glycaemic effects [25].

Reviews recommend monitored dairy reduction, prioritising low-sugar alternatives while maintaining micronutrient sufficiency [1,18].

Clinical interpretation:

Given current evidence, a cautious, personalised trial of dairy reduction—especially of low-fat, sweetened milk beverages—may be reasonable, with attention to nutrient sufficiency.

Dietary Fat Composition: SFA, MUFA, PUFA

Western dietary patterns high in saturated fat (SFA) promote inflammatory metabolic states associated with acne severity [25,26]. Sebum enriched in SFA is more prone to lipid peroxidation, intensifying local inflammatory cascades [4].

In contrast:

MUFA (olive oil) support antioxidant defence and lipid homeostasis

Omega-3 PUFA lower production of pro-inflammatory eicosanoids [22,24]

Given this contrast, dietary models rich in high-quality unsaturated fats — particularly MD — demonstrate lower acne burden [11–13].

Clinical interpretation:

Dietary recommendations should reduce SFA and increase MUFA and omega-3 PUFA intake.

Gluten-Containing Foods and Gluten-Free Dietary Patterns

A Mendelian randomisation analysis found no causal link between gluten-free diet and acne risk [27]. Clinical recommendations state that gluten restriction is unjustified unless gluten-related disease is present [32].

Reports of improvement after reducing wheat products likely reflect lower glycaemic exposure, not gluten removal itself [25,29]. Surveys confirm that gluten avoidance is commonly attempted, but lacks evidence-based benefit [17].

Clinical interpretation:

Do not recommend gluten-free diets for acne in the absence of gluten-related disorders. Focus on lowering glycaemic load (e.g., replacing refined wheat products with minimally processed, higher-fibre alternatives).

Note on intestinal mechanisms

Emerging research suggests a potential influence of intestinal physiology on systemic inflammation relevant to acne. However, the detailed evaluation of the gut microbiome or targeted microbiome-modulating strategies exceeds the scope of this review, which focuses strictly on dietary factors with the strongest mechanistic and clinical relevance to acne vulgaris [15,16,18].

Discussion

This narrative review synthesised evidence from 32 clinical, mechanistic, observational and consensus publications on dietary influences in acne vulgaris. Across study types, diet-related exposures were linked to pathways considered relevant to acne biology, including insulin–IGF-1–mTORC1 signalling, inflammatory cytokine expression, lipid biosynthesis and oxidative stress [1,4–6]. These mechanistic observations support the plausibility of diet as an adjunctive target; however, they do not by themselves establish clinical efficacy.

Among dietary models, the Mediterranean diet (MD) shows reproducible associations with lower acne severity in observational settings [11–14,18–21]. By contrast, low-glycaemic-load (LGL) approaches have the most actionable clinical signal, with benefits suggested by interventional and observational data, including at least one randomised trial [8–10]. Conceptually, MD may confer benefit via a nutrient pattern rich in antioxidants, polyphenols and unsaturated fats, whereas LGL interventions act more directly on postprandial insulin dynamics—currently the most substantiated dietary driver of acne progression. Importantly, the MD signal remains largely non-randomised, and effect sizes across studies are heterogeneous.

Westernised patterns high in refined sugars, high-GL foods and sweetened dairy products are consistently associated with higher acne burden across different populations [9,10,25,26,29,31]. While the cross-cultural reproducibility of these findings strengthens biological credibility, residual confounding (e.g., broader lifestyle factors, under-reporting, weight status) cannot be excluded.

Evidence around plant-forward eating indicates that potential benefits are most likely when emphasis is placed on whole-food quality and lower glycaemic load; veganism per se is not supported as a therapeutic strategy [22–27]. Very low-calorie ketogenic diets have mechanistic rationale but remain experimental in acne due to a lack of condition-specific trials [18,28]. Routine gluten restriction is not justified without coexisting gluten-related pathology; reported improvements after reducing wheat products probably reflect reductions in glycaemic load rather than gluten per se [27,32].

From a care-delivery perspective, nearly half of patients with moderate-to-severe acne report self-directed dietary changes [17]. This underscores both the demand for guidance and the risk of unsupervised, restrictive patterns. Embedding structured, individualised nutrition counselling into dermatological care may help translate the current, mostly associative evidence into safer, patient-centred practice while we await higher-quality trials.

Strengths and limitations

Strengths of this review include integration of mechanistic and clinical data, parallel consideration of whole-diet patterns and specific food groups, and a focus on clinically actionable themes (notably glycaemic load and fat quality).

Limitations mirror the underlying literature: most studies are cross-sectional or case–control with self-reported diet, variable acne grading and short follow-up; 6 tandardiz evidence for whole-diet models (particularly MD, plant-based and ketogenic strategies) is scarce. Confounding by overall diet quality, energy intake, BMI, medication use and broader lifestyle factors remains a concern, and publication bias cannot be excluded. Future research should prioritise adequately powered, 6tandardiz, whole-diet interventions with 6tandardized outcome measures, predefined biomarkers (e.g., IGF-1) and longer follow-up to clarify effect sizes and identify subgroups most likely to benefit.

Conclusions

Available studies suggest that dietary patterns can influence acne biology and clinical expression. Low-glycaemic-load diets show the most actionable evidence, whereas associations with Mediterranean-style eating are encouraging but mainly observational. Diets high in refined sugars, high-GL foods and sweetened dairy consistently track with higher acne severity. In the absence of gluten-related pathology, gluten restriction lacks rationale.

Given current evidence—largely observational with few randomised trials—dietary change should be considered as a supportive measure alongside standard dermatological care. Structured, individualised nutrition counselling may aid symptom control and quality of life, while rigorous, adequately powered trials of whole-diet interventions remain a priority.

Disclosures

Authors' contributions: conceptualization, Wiktor Kubik; methodology, Bartłomiej Czarnecki, Jan Nowak, and Illia Koval; software, Bartosz Zwoliński; check, Bartosz Zwoliński and Katarzyna Szewczyk; formal analysis, Bartłomiej Czarnecki, Jan Nowak, and Illia Koval; investigation, Wiktor Kubik, Wirginia Bertman, Natalia Kołdej, Klaudia Romejko; resources, Kacper Sukiennicki; data curation, Bartosz Zwoliński, Zuzanna Kępczyńska, Kamil Borysewicz; writing – rough preparation, Wiktor Kubik, Kacper Sukiennicki, Klaudia Romejko, Kamil Borysewicz; writing – review and editing, Wirginia Bertman, Natalia Kołdej; supervision, Zuzanna Kępczyńska, Katarzyna Szewczyk; project administration, Bartłomiej Czarnecki, Katarzyna Szewczyk.

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