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THE ROLE OF DIET IN THE PATHOGENESIS AND TREATMENT OF ACNE VULGARIS

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ABSTRACT

Background: Acne vulgaris is a common chronic inflammatory skin condition affecting the pilosebaceous unit. Although hormonal factors are well-recognized in its pathophysiology, emerging evidence suggests that dietary factors may influence the onset, severity, and progression of acne.
Objective: This review aims to evaluate the current literature on the role of diet in acne vulgaris, focusing on specific dietary components that may exacerbate or alleviate the condition.
Methods: A literature review was conducted to examine the relationship between dietary patterns—including glycemic index, dairy intake, dietary fats, and micronutrients—and acne pathogenesis. The biological mechanisms through which these factors may influence sebaceous activity, inflammation, and hormonal regulation were also explored. Databases such as PubMed and Google Scholar were searched using the following terms: "acne vulgaris," "IGF-1 and acne," "mTORC1 acne pathway," and "inflammatory skin diseases." Studies were selected based on their relevance to dietary modulation of acne pathophysiology, mechanistic insights, and clinical outcomes.
Results: High-glycemic index diets, dairy consumption, and saturated and trans fats have been associated with increased acne severity, potentially through the stimulation of insulin and IGF-1 (nsulin-like growth factor-1) pathways, activation of mTORC1, and upregulation of sebum production. Conversely, diets low in glycemic load and rich in omega-3 fatty acids may have protective effects by reducing inflammation and normalizing sebaceous gland activity.
Conclusion: Diet appears to play a significant role in acne pathophysiology through various hormonal and inflammatory mechanisms. While further large-scale and controlled studies are needed, dietary modification may serve as a useful adjunct in acne management.

KEYWORDS

Acne Vulgaris, Inflammatory Skin Diseases, Glycemic Index, Comedogenesis, Sebum Production, Nutritional Dermatology

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Introduction.

Acne vulgaris is a chronic inflammation of the pilosebaceous unit, primarily affecting adolescents, with an estimated lifetime prevalence of up to 85% (Bhate & Williams, 2012). Its onset is strongly correlated with hormonal changes that occur throughout life. Although acne most commonly arises during adolescence, it may persist into adulthood, with prevalence generally declining after the third decade of life (Heng & Chew, 2020).

Multiple factors contribute to the pathogenesis of acne, leading to the formation of the primary lesion known as a comedo. The face, chest, and upper back are the most commonly affected areas. In moderate to severe cases, acne can result in post-inflammatory hyperpigmentation and permanent scarring. Severe manifestations of the disease are often associated with significant psychosocial consequences; individuals with acne have been shown to be at increased risk for anxiety, depression, and reduced quality of life (Williams, Dellavalle & Garner, 2011).

Effective treatment requires a multifaceted approach that addresses the underlying pathophysiological mechanisms as well as patient-specific factors. While hormonal influences are well established, increasing attention has turned toward the potential role of diet in the development and severity of acne. This review aims to examine the current evidence on dietary factors and their relationship to acne vulgaris.

Pathophysiology

Acne lesions are classified into two main categories: (1) non-inflammatory lesions, such as open and closed comedones, and (2) inflammatory lesions, including papules, pustules, nodules, cysts, and abscesses. These lesions may resolve with varying degrees of post-inflammatory hyperpigmentation and scarring (Vasam, Korutla & Bohara, 2023)

Four principal mechanisms contribute to acne pathogenesis:

1. Follicular hyperproliferation and abnormal keratinization of the infundibulum, leading to comedone formation;

2. Increased sebum production and retention, influenced by elevated levels of androgens, particularly dihydrotestosterone (DHT);

3. Colonization by *Cutibacterium acnes* (formerly *Propionibacterium acnes*), which promotes inflammation;

4. Activation of innate and adaptive inflammatory pathways, contributing to lesion progression and tissue damage (Zaenglein, 2018).

Established risk factors for acne include genetic predisposition, family history, polycystic ovary syndrome (PCOS), metabolic syndrome, and environmental influences such as smoking and the use of comedogenic skincare products (Tan, Schlosser & Paller, 2017)

Emerging evidence suggests that diet may act as a modifiable risk factor by contributing to the synthesis of sebaceous lipids. This thesis is supported by the observation that linoleic acid, a substrate of sebum, must be provided through the diet. It has been shown that increased consumption of dietary fats and carbohydrates alters the lipid composition and increases sebum production (Smith et al.., 2008).

Individuals following a Western diet—characterized by a high intake of processed and high-fat foods, refined sugars, dairy, and red meat—are more prone to developing acne, as this dietary pattern has a potentiating effect on serum insulin and insulin-like growth factor-1 (IGF-1) levels (Wu et al.., 2020; Zouboulis, 2020).

The research group of Deplewski and Rosenfield established that the IGF-1 levels, which peak during puberty correlate with the clinical manifestation of acne to a higher extent than the levels of serum androgens (Deplewski & Rosenfield, 1999). Furthermore, elevated serum levels of Insulin Growth Factor-1 (IGF-1) have been observed in both male and female patients with acne. A correlation has been shown between serum IGF-1 levels, acne lesions, 5α-DHT, and DHEA sulfate (Makrantonaki, Ganceviciene & Zouboulis, 2011).

IGF-1 stimulates sebofollicular androgen receptors in two ways: (1) by increasing gonadal and adrenal androgen synthesis and promoting the conversion of testosterone to DHT, and (2) by activating androgen receptors through FoxO1 suppression. Suppression of FoxO1 and activation of mTORC1 by dietary components may increase sebum production and alter sebum composition, promoting *Propionibacterium acnes* overgrowth and inflammation (Melnik, 2015).

Insulin Growth Factor-1 (IGF1) also influences the epidermal homeostasis. IGF-1 receptors have been found in the maturing sebocytes and suprabasal duct cells suggesting its role as a sebaceus mitogen and morphogen (Melnik & Schmitz, 2009). IGF-1 induces lipid synthesis in human sebocytes by stimulating sterol regulatory element-binding protein-1 (SREBP-1) (Makrantonaki, Ganceviciene & Zouboulis, 2011). An interaction between IGF-1 and estradiol levels has been described, implying a possible link between estradiol levels and acne pathogenesis (Makrantonaki et al.., 2008).

Kim et al.. (2017) concluded that IGF-1 increases the expression of inflammatory biomarkers (IL-1β, IL-6, IL-8, TNF-α) in cultured sebocytes, thereby increasing sebum production.

Key dietary elements further reviewed in this article include: high glycemic index (GI) foods, dairy products, dietary fats (particularly saturated facts and polyunsaturated fatty acids). These categories will be examined in detail in the following sections.

Key dietary factors and their effect on acne

Glycemic Index

Glycemic index (GI), reflects the rise in blood glucose levels after injesting certain foods and beverages. It is measured on a scale from 1 to 100 reflecting the rate at which carbohydrate is metabolized in comparison to standard amount of glucose. High GI foods (>70) increase the blood glucose level more rapidly resulting in higher spikes, compared to Low GI foods (<55), having a more gradual rise (Barclay et al.., 2021). Diets rich in high-GI carbohydrates have been shown to increase IGF-1 levels, a hormone that affects lipogenesis and androgen metabolism (Kim et al.., 2017). In contrary, low glycemic index diets have been shown to reduce IGF-1 levels (Burris et al.., 2018). A trial conducted by Smith et al.. underlined that a low-GI diet increases

insulin sensitivity. The authors found that patients with acne demonstrated hyperinsulinemia during an oral glucose tolerance test. Increased insulin sensitivity may lead to a reduction in testosterone bioavailability and DHEA concentration, resulting in decreased acne severity (Smith et al.., 2007).

A randomized study involving patients with mild to severe acne found that adherence to a low glycemic load (GL) diet led to a reduction in both non-inflammatory and inflammatory acne lesions, along with decreased sebaceous gland activity and acne severity grade (Kwon et al.., 2012). A systematic review by Meixiong et al.. (2022) concluded that a high-GI diet may exacerbate acne, whereas adherence to a low-GI diet may reduce acne lesion counts and severity.

High-GI and high-GL diets alter the composition of sebum fatty acids, promoting a proinflammatory and comedogenic effect in acne pathogenesis. Carbohydrate reduction through decreased insulin signaling increases nuclear FoxO1 and attenuates mTORC1 activity, leading to reduced expression of SREBP-1. This results in decreased sebum triglyceride fatty acid desaturation and reduced total sebum production (Melnik, 2015).

Low glycemic load has also been shown to predict a positive response to isotretinoin treatment, with no significant difference observed in the efficacy of topical benzoyl peroxide 2.5% (Baldwin & Tan, 2020).

Diary

Dairy, particularly cow's milk, is one of the main sources of protein in the Western diet. Analysis of its micronutrients shows that cow's milk contains casein (approximately 80% of total protein) and whey proteins (20%), including α-lactalbumin and β-lactalbumin (Podgórska et al.., 2021). Casein is reported to stimulate IGF-1 to a greater extent than whey proteins, whereas whey proteins have an insulinotropic effect. Whey proteins contain six growth factors that stimulate pancreatic insulin secretion: fibroblast growth factor-1 (FGF-1), FGF-2, IGF-1, IGF-2, platelet-derived growth factor (PDGF), and transforming growth factor (TGF) (Ryguła, Pikiewicz & Kaminiów, 2024).

Milk products also contain other bioactive substances, including hormone precursors, IGF-1, and TGF-β. They are rich in branched-chain amino acids (leucine, isoleucine, valine), which stimulate insulin secretion and raise both insulin and IGF-1 levels, mimicking the effects of high-GI foods (Podgórska et al.., 2021). Additionally, milk enhances the FOXO1/mTORC1/SREBP1c pathway, enhancing sebaceous gland hypertrophy and lipogenesis (Ryguła, Pikiewicz & Kaminiów, 2024).

Research indicates that the acnegenic potential of milk is strongly linked to its hormonal and bioactive components rather than its fat content. Even though milk and unsweetened dairy products have a low glycemic index, their hormonal composition affects insulin and IGF-1 levels similarly to high-GI foods (Dall'Oglio et al.., 2021). Industrial milk has also been reported to contain anabolic steroids and other growth factors, which may promote hyperkeratinization of the pilosebaceous unit and increased sebum production (Claudel et al.., 2018).

LaRosa et al.. (2016) found a positive association between the consumption of skimmed or low-fat milk and acne. This may result from food processing increasing the bioavailability of acne-promoting triggers. Another hypothesis suggests that skimmed milk contains lower levels of estrogen, which otherwise has a protective effect on acne lesions (Adebamowo et al.., 2004).

A systematic review and meta-analysis by Juhl et al.. (2018) concluded that the intake of any dairy—milk or yogurt—was associated with a higher odds ratio of acne in individuals aged 7–30 years, regardless of the amount or frequency. Cheese intake was associated with a borderline increase in risk.

The NutriNet-Santé cohort study also demonstrated an association between milk consumption and the presence of acne. The analysis showed that individuals with acne consumed significantly more milk, milk chocolate, and other components typical of a Western diet (Penso et al.., 2020).

Whey Protein Supplementation

Whey protein and its supplements—often consumed to increase muscle mass—have been linked to acne. The comedogenic effect of whey protein is thought to result from increased IGF-1 synthesis, leading to hyperproliferation and excess sebum production (Cava et al.., 2024).

Research conducted by Cava et al.. (2024) highlighted the connection between whey protein intake and acne progression. Human studies reviewed in their analysis consistently found that whey protein supplementation exacerbated acne, while discontinuation led to improvements in skin condition.

Fatty acids

Saturated FA

The Western diet is characterized by high levels of saturated fatty acids, which may serve as acne triggers (Ryguła, Pikiewicz & Kaminiów, 2024). Saturated fatty acids—especially palmitate—are proinflammatory, acting through mTORC1 activation and promoting keratinocyte proliferation, leading to comedogenesis. Palmitate accounts for 32% of milk triglycerides, reinforcing the theory of milk's acne-promoting potential. Industrially produced trans-fatty acids, commonly found in fast food, structurally resemble palmitate and are also associated with acne via TLR2/TLR4 activation (Melnik, 2015).

Supporting this hypothesis, Burris, Rietkerk, and Woolf (2014) demonstrated in a cohort study of young adults in New York that increased intake of saturated fats and trans fats correlated with acne severity. Similarly, the NutriNet-Santé Prospective Cohort Study found that energy-dense dietary patterns high in fat and sugar exacerbated acne lesions (Penso et al.., 2020).

Unsaturated Fatty Acids

Omega-3 fatty acids (ω-3 FAs) cannot be synthesized by the human body and must be obtained from the diet or supplementation. They may be derived from plant/algae sources or fish. Plant-based ω-3 FAs are primarily in the form of alpha-linolenic acid (ALA), which must be metabolized into the more biologically active forms eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Algae and fish are rich in EPA and DHA directly.

Among their many beneficial properties, ω-3 FAs exert anti-inflammatory effects by promoting the production of prostaglandins (PGE1, PGE3) and leukotriene B5, while also downregulating IGF-1. This reduces sebum production and hyperkeratinization of the pilosebaceous unit (Guertler et al.., 2024).

In Western diets, the ω-3 to ω-6 FA ratio is significantly lower due to insufficient intake of fish, wild game, and plant-based sources. ω-6 FAs, such as arachidonic acid, may worsen acne by promoting inflammatory cytokines (IL-6, IL-8) and stimulating keratinocyte proliferation. Additionally, monounsaturated fatty acids have been correlated with increased sebum production and acne severity (Liu et al.., 2025).

A pilot study by Guertler et al.. (2024) found that all acne patients, regardless of clinical severity, had ω-3 FA deficiencies—with the most severe deficits observed in male participants. These findings support the hypothesis that ω-3 FA deficiency may play a role in acne exacerbation.

In patients receiving oral isotretinoin therapy, omega-3 supplementation may also help reduce dermatological side effects such as cheilitis, xerosis, and dryness of the eyes and nose (Mirnezami & Rahimi, 2018).

A randomized controlled trial by Jung et al.. (2014) examined the impact of omega-3 or γ-linoleic acid supplementation over 10 weeks in 45 patients with mild-to-moderate acne. Supplementation led to a reduction in both inflammatory and non-inflammatory acne lesions.

Discussion

This review highlights the significant role dietary factors may play in the pathogenesis and progression of acne vulgaris. While acne has a strong link to hormonal changes, sebaceous hyperactivity, and bacterial colonization, growing evidence suggests the importance of nutrition.

Among the most consistently supported findings is the association between high glycemic index (GI) diets and increased acne severity. High-GI foods promote hyperinsulinemia, which—through elevated IGF-1 levels and activation of the mTORC1 pathway—enhances sebocyte activity and inflammatory cytokine production (Kim et al., 2017). In contrast, low-GI diets have been shown to improve acne severity through the reduction of both inflammatory and non-inflammatory acne lesions (Meixiong et al., 2022). These findings highlight the potential benefits of glycemic control in acne management, particularly in patients with insulin resistance or metabolic syndrome.

Although dairy products have a low glycemic index, their hormonal and bioactive content, including IGF-1 and insulinotropic amino acids, mimics the endocrinological effects of high-GI foods (Podgórska et al., 2021; Ryguła et al., 2024). Industrial milk may contain growth-promoting hormones and steroids that further exacerbate acne through androgenic and comedogenic pathways (Claudel et al., 2018). Epidemiological studies have consistently shown an association between dairy intake and acne prevalence, particularly in adolescents and young adults (Juhl et al., 2018; LaRosa et al., 2016; Penso et al., 2020), although causation cannot be firmly established.

Fatty acid composition also appears to be linked to acne exacerbation. Diets high in saturated and trans fats may lead to increased acne severity through pro-inflammatory effects and stimulation of keratinocyte

proliferation via mTORC1 activation (Melnik, 2015). In contrast, omega-3 fatty acids may help normalize sebaceous gland activity by downregulating IGF-1 and inflammatory cytokines (Guertler et al., 2024). Although evidence remains preliminary, omega-3 supplementation could serve as an adjunctive therapy, especially in patients with documented dietary deficiencies or those undergoing isotretinoin treatment (Mirnezami & Rahimi, 2018).

Despite the promising associations between diet and acne, several limitations should be acknowledged. Most of the current evidence is derived from observational studies, which are subject to bias. Dietary intake is difficult to quantify precisely, and studies often vary in methodology, sample size, and outcome assessment. Moreover, genetic and hormonal factors may moderate the dietary influence on acne, suggesting that dietary recommendations should be individualized rather than universally applied.

Conclusions

Acne vulgaris is a multifactorial disease influenced by genetic, hormonal, environmental, and increasingly recognized dietary factors. Despite these promising associations, it is important to acknowledge that diet alone is unlikely to serve as a standalone treatment for acne. This review highlights the complex interplay between nutrition and acne pathogenesis. High-glycemic diets, dairy products—particularly skimmed milk—and certain fats may exacerbate acne through hormonal and inflammatory pathways, primarily involving insulin, IGF-1, and mTORC1 activation. In contrast, low-GI diets and increased intake of omega-3 fatty acids may offer anti-inflammatory and sebum-regulating benefits.

Although dietary interventions may not replace standard pharmacologic treatments, they represent a promising complementary approach. Future research should focus on large-scale randomized controlled trials and mechanistic studies to further define the role of diet in acne and establish evidence-based dietary guidelines for clinical practice.

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