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Review Article

Dietary Modulation of the NLRP3 Inflammasome in Inflammatory Skin Disease: A Targeted Review

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Abstract

The NOD-like receptor protein 3 (NLRP3) inflammasome plays a central role in the pathogenesis of numerous inflammatory skin diseases, including psoriasis, atopic dermatitis, acne, and hidradenitis suppurativa. Emerging evidence suggests that dietary factors can significantly modulate NLRP3 activation through pathways involving oxidative stress, mitochondrial dysfunction, toll-like receptors, and cytokine regulation. This review synthesizes findings from in vitro, in vivo, and clinical research studies, evaluating the influence of specific nutrients and dietary patterns on NLRP3 activity in skin-related contexts. Compounds such as omega-3 fatty acids, vitamin D, polyphenols, and flavonoids consistently demonstrated inhibitory effects on NLRP3 inflammasome activation, while Western dietary patterns, saturated fats, and hyperglycemic states were associated with its upregulation. Mechanistic insights across studies revealed modulation of IL-1β, IL-18, ROS, ASC speck formation, and autophagy as key regulatory nodes. Translational findings highlight the potential for dietary interventions to complement pharmacologic therapies and mitigate chronic skin inflammation through targeted inflammasome suppression. By elucidating diet-inflammasome-skin interactions, this review supports the integration of nutritional strategies into the management of inflammatory dermatoses and offers a foundation for future interventional research.

Abbreviations

AD: Atopic Dermatitis; AI: Artificial Intelligence; AMPK: AMP-Activated Protein Kinase; ASC: Apoptosis-Associated Speck-like Protein Containing a Caspase Recruitment Domain; BHB: β-Hydroxybutyrate; DAMP: Damage-Associated Molecular Pattern; DHA: Docosahexaenoic Acid; EPA: Eicosapentaenoic Acid; HDACi: Histone Deacetylase Inhibitor; HS: Hidradenitis Suppurativa; IL: Interleukin; LDL: Low-Density Lipoprotein; LY: Low-Yield; NAD+: Nicotinamide Adenine Dinucleotide; NF-κB: Nuclear Factor Kappa B; NLRP3: NOD-Like Receptor Protein 3; Nrf2: Nuclear Factor Erythroid 2–Related Factor 2; PAMP: Pathogen-Associated Molecular Pattern; PKC: Protein Kinase C; PP2A: Protein Phosphatase 2A; RD: Registered Dietitian; ROS: Reactive Oxygen Species; SFA: Saturated Fatty Acid; SIRT1: Silent Information Regulator Sirtuin 1; SCFA: Short-Chain Fatty Acid; Th: T Helper; TLR: Toll-Like Receptor; ULK1: Unc-51-Like Kinase 1; VDR: Vitamin D Receptor; VLCKD: Very Low-Calorie Ketogenic Diet; WD: Western Diet

Introduction

The NLRP3 inflammasome is a cytosolic multiprotein signaling complex that senses a variety of cellular stress signals, including pathogen-associated molecular patterns (PAMPs), damage-associated molecular patterns (DAMPs), and metabolic danger signals. Structurally, it comprises the NOD-like receptor NLRP3, the adaptor protein ASC (apoptosis-associated speck-like protein containing a caspase

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recruitment domain), and the effector enzyme pro-caspase-1. Upon activation, the inflammasome facilitates caspase-1-dependent cleavage of the pro-inflammatory cytokines pro-IL-1β and pro-IL-18 into their mature, secreted forms, thereby amplifying local and systemic inflammatory responses [1,2]. Additionally, inflammasome activation can trigger a form of lytic programmed cell death known as pyroptosis, which is mediated by gasdermin D pore formation and further contributes to immune cell recruitment and tissue damage [3,4]. In the skin, NLRP3 activation plays a critical role in the pathogenesis of several chronic inflammatory dermatoses. Psoriatic lesions demonstrate upregulation of inflammasome-related transcripts and proteins, including NLRP3, ASC, and caspase-1, in keratinocytes and immune infiltrates [5,6]. In acne vulgaris, Cutibacterium acnes activates NLRP3 in sebocytes and macrophages, promoting IL-1β secretion and comedogenesis [7,8]. Hidradenitis suppurativa has also been linked to elevated expression of inflammasome-related genes in lesional skin, potentially contributing to chronic neutrophilic inflammation [9,10]. In atopic dermatitis, emerging evidence suggests that dysregulated NLRP3 signaling in macrophages and keratinocytes contributes to epithelial barrier disruption and Th2-skewed inflammation [11,12]. A growing body of literature supports a functional diet-inflammation-skin axis. Specific dietary components, such as omega-3 polyunsaturated fatty acids, polyphenols, genistein, curcumin, and fasting-derived metabolites, have been shown to inhibit NLRP3 activation via mechanisms including suppression of mitochondrial reactive oxygen species (ROS) production, blockade of NF-κB translocation, and enhancement of autophagy [13-17]. Conversely, Western-style dietary patterns high in saturated fats, refined sugars, and inflammatory lipids have been associated with enhanced NLRP3 activation and worsening of inflammatory phenotypes [18-21]. While prior reviews have explored dietary patterns and general inflammation, few have specifically examined the molecular interface between dietary modulation and NLRP3 inflammasome activity in the context of skin disease. This review aims to fill that gap by synthesizing mechanistic findings from preclinical, translational, and clinical studies that investigate how diet influences inflammasome-driven cutaneous inflammation.

Methods

This targeted review was conducted through a comprehensive literature search using PubMed, Scopus, and Web of Science. The search strategy employed Boolean combinations of terms such as "NLRP3 inflammasome," "dietary modulation," "nutrition," "IL-1β," "IL-18," "skin inflammation," "psoriasis," "acne," "atopic dermatitis," and "hidradenitis suppurativa." Studies were included if they were published between January 2010 and May 2025, investigated the modulation of the NLRP3 inflammasome by dietary compounds or patterns, and used in vitro, animal, or human models relevant to cutaneous or immunologic inflammation. Articles were required to report measurable outcomes related to NLRP3 activation or downstream mediators, including IL-1β, IL-18, caspase-1, ASC, or ROS. Studies were excluded if they focused on non-dietary triggers of NLRP3 activation (such as microbial or mechanical stimuli), lacked involvement of the NLRP3 inflammasome pathway, or were centered on non-dermatologic systems such as cardiovascular or neurologic models. Additional exclusion criteria included non-English publications and studies without primary data. Relevant data from each included study were extracted and categorized by study type, nutrient class (e.g., omega-3 fatty acids, polyphenols, vitamins, fasting metabolites), and disease relevance (e.g., psoriasis, acne, atopic dermatitis, hidradenitis suppurativa, or wound healing) to facilitate pattern recognition in inflammasome modulation.

NLRP3 and inflammatory skin diseases

The NLRP3 inflammasome plays a pivotal role in the pathogenesis of various inflammatory skin diseases, including psoriasis, acne, hidradenitis suppurativa (HS), and atopic dermatitis (AD). Its activation leads to the release of pro-inflammatory cytokines such as interleukin-1β (IL-1β) and interleukin-18 (IL-18), contributing to disease progression and symptom severity.

Psoriasis: The NLRP3 inflammasome plays an important role in the pathogenesis of psoriasis, a chronic inflammatory skin disease marked by keratinocyte hyperproliferation and immune dysregulation. In vitro and in vivo studies have demonstrated that NLRP3 directly promotes keratinocyte proliferation and inflammatory cytokine expression, key contributors to the pathogenesis of psoriasis [22]. Notably, in psoriatic skin biopsy specimens, elevated expression of NLRP3, caspase-1, and IL-1β has been observed, with levels up to four times higher compared to controls [23,24]. This overactivation leads to increased caspase-1-mediated cleavage of pro-inflammatory cytokines IL-1β and IL-18, both of which are vital contributors to inflammation associated with psoriasis [25]. The levels of these cytokines in circulation are also significantly elevated in untreated psoriasis patients, further suggesting their role in disease severity and systemic manifestations [23]. Furthermore, given its critical role in disease development, dietary modulation of NLRP3 activity presents a promising approach to the management of psoriasis.

Acne: The presence of Propionibacterium acnes (P. acnes) plays a critical role in the pathophysiology of acne vulgaris, initiating an immune response largely mediated by the NLRP3 inflammasome. Specifically, both sebocytes and monocytes respond to P. acnes exposure with heightened levels of IL-1β, driven by NLRP3 and caspase-1 activity [26,27]. Notably, knockdown of NLRP3 in sebocytes or monocytes markedly reduces IL-1β production, thus emphasizing the requirement of this pathway for inflammasome activation [26,27]. In vivo, when NLRP-3-deficient mice are injected with P. acnes, they show diminished inflammation levels compared to controls, and this response is similar to mice in which IL-1β is disrupted [26,28]. This confirms the key role of NLRP-3 in the inflammatory pathogenesis of acne. These findings thus establish the NLRP-3 inflammasome as a promising target for managing acne symptoms, potentially through dietary modulation.

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Hidradenitis suppurativa: The NLRP3 inflammasome plays a key role in the pathophysiology of HS, often contributing to follicular rupture and chronic inflammation. Levels of NLRP3 genes are notably heightened in the visibly affected and surrounding skin, although levels were significantly higher in lesional versus non-lesional skin [29]. This suggests NLRP3's role in the spread of associated inflammation. The NLRP3 inflammatory pathway was particularly active in resident immune cells (such as dendritic and Langerhans cells), where it promotes the secretion of IL-1β and IL-17A – key drivers of inflammation and lesion formation [20]. As inflammation progresses and hyperproliferation occurs, the follicular wall ruptures, releasing intracellular components that further inflammasome activation, which are sensed by keratinocytes and immune cells, which amplify the release of more inflammatory components [30]. This ongoing cycle manifests as chronic inflammation in HS patients. Notably, blocking NLRP3 in HS skin significantly reduces the production of these inflammatory mediators, confirming its role in maintaining this cycle [20]. Taken together, this makes NLRP3 a great potential therapeutic target for managing HS through dietary decisions.

Atopic dermatitis: Compromise of the skin barrier is considered an early and foundational event in the onset of AD [31]. Recent findings suggest that NLRP3 inflammasome activation may contribute to this barrier dysfunction through both direct and indirect mechanisms. Specifically, it was shown that inhibiting the NLRP3 pathway with mdivi-1 in a mouse model reduced the levels of NLRP3 and IL1β and IL-18 significantly, while also reducing features characteristic of AD [32]. This points to the potential role of NLRP3 in the inflammatory pathogenesis of AD. Beyond this, NLRP3 may also influence skin barrier integrity. This was proposed specifically because NLRP3 has been shown to regulate Th1 and Th22 cytokine expression, which are known to downregulate key structural proteins that make up a strong skin barrier [33]. These data thus point to a role of NLRP3 in exacerbating skin barrier compromise to further enhance the progression of AD, ultimately pointing to dietary modulation of NLRP3 as a potential strategy in mitigating AD symptoms.

Dietary modulation of NLRP3: Pro- and anti-inflammatory factors

Emerging evidence suggests that dietary choices play a critical role in either promoting or suppressing NLRP3 inflammasome activity in the skin (Table 1). The NLRP3 inflammasome acts as a molecular sensor that responds to danger signals within the body, including those triggered by poor dietary habits [34]. Nutrients and dietary patterns influence key upstream processes such as mitochondrial oxidative stress, cytokine production, epithelial barrier integrity, and gut microbiota composition, all of which can either activate or inhibit inflammasome assembly [35]. From a mechanistic standpoint, pro-inflammatory diets are known to increase levels of ROS, activate toll-like receptors (TLRs), and disrupt metabolic homeostasis, all of which contribute to the activation of NLRP3 and subsequent release of inflammatory cytokines like IL-1β and IL-18 [36]. Conversely, anti-inflammatory nutrients and dietary strategies appear to modulate these pathways by enhancing antioxidant defense systems, supporting autophagy, and restoring immune balance [37]. Understanding the relationship between diet and the inflammasome provides a unique opportunity for clinicians and registered dietitians to address chronic skin inflammation through targeted nutritional interventions. Inflammatory skin diseases such as psoriasis, acne, hidradenitis suppurativa, and atopic dermatitis are increasingly being recognized as systemic conditions influenced not only by immune dysregulation and genetics, but also by modifiable lifestyle factors, including diet. This section explores both pro-inflammatory and anti-inflammatory dietary triggers and their impact on NLRP3 inflammasome activation, with the aim of supporting integrative strategies in dermatologic care.

Pro-inflammatory triggers: Certain dietary components have been shown to act as pro-inflammatory stimuli that can prime and activate the NLRP3 inflammasome, thereby

Table 1: Dietary Modulators of NLRP3 Inflammasome Activation and Their Mechanisms in Skin Disease.

Dietary Modulator	Source(s)	Mechanism of Action	Net Effect on NLRP3	Skin Disease(s)
Omega-3 Fatty Acids (EPA/DHA)	Fatty fish, flaxseed, chia	Inhibits NF-κB, reduces ROS, alters membrane fluidity	↓ Suppression	Psoriasis, AD
Polyphenols (e.g., resveratrol, curcumin, EGCG)	Grapes, turmeric, green tea	Suppresses NF-κB, reduces ROS, inhibits NLRP3 assembly	↓ Suppression	Psoriasis, Acne
Vitamin D (Calcitriol)	Sunlight, fortified foods, supplements	Modulates VDR, suppresses NLRP3 gene expression, reduces IL-1β	↓ Suppression	Psoriasis, AD
β-Hydroxybutyrate (Ketogenic/Fasting Diets)	Endogenous (fasting, ketogenic diet)	Direct NLRP3 inhibition, autophagy induction, mitochondrial stabilization	↓ Suppression	Psoriasis, HS, AD
Fiber/SCFAs (Butyrate)	Whole grains, fruits, vegetables	Gut microbiome fermentation; inhibits HDAC, suppresses NF-κB	↓ Suppression	Psoriasis, AD
Saturated/Trans Fats	Processed meats, dairy, fried foods	Induces TLRs, increases ROS, activates caspase-1, primes NLRP3	↑ Activation	All
Refined Sugars/High GI Foods	White bread, soda, sweets	Glycemic/insulin spikes, oxidative stress, cytokine upregulation	↑ Activation	Acne, Psoriasis
Western Dietary Pattern	High in fats, sugars, processed foods	Chronic metaflammation, gene upregulation, microbiome disruption	↑ Activation	All

Abbreviations: EPA: Eicosapentaenoic acid; DHA: Docosahexaenoic acid; NF-κB: Nuclear Factor Kappa B; ROS: Reactive Oxygen Species; VDR: Vitamin D Receptor; AD: Atopic Dermatitis; HS: Hidradenitis Suppurativa; SCFA: Short Chain Fatty Acid; GI: Glycemic Index.

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contributing to chronic skin inflammation [17,38]. These triggers initiate processes such as mitochondrial dysfunction, increased ROS, and enhanced cytokine production, all of which serve to amplify the inflammatory cascade in dermatoses such as psoriasis, acne, hidradenitis suppurativa, and atopic dermatitis.

Saturated fats and trans fats: These fats are potent dietary activators of the NLRP3 inflammasome. Saturated fatty acids, particularly palmitic acid, can activate TLRs, promote mitochondrial ROS production, and initiate caspase-1–dependent IL-1β and IL-18 secretion [39-41]. Dietary sources include fatty cuts of red meat (e.g., ribeye, ground beef), processed meats (sausages, bacon), full-fat dairy (milk, cheese, butter), commercial baked goods, fast food, and deep-fried items. Trans fats, found in partially hydrogenated oils and processed snacks, are also associated with elevated systemic inflammation and skin flare-ups.

High glycemic index (GI) and glycemic load (GL) foods: Foods that rapidly spike blood glucose levels stimulate insulin and subsequently increase levels of pro-inflammatory cytokines. This glycemic volatility contributes to oxidative stress and inflammasome priming [42,43]. Common examples include white bread, sweetened cereals, pastries, sugary beverages (like soda or juice), and candy. Frequent consumption of these items is linked to worsened acne severity and greater psoriatic plaque activity, likely via elevated IL-1β signaling.

Excessive fructose and refined carbohydrates: Fructose-rich foods, especially those containing high-fructose corn syrup (found in soda, sweetened condiments, and packaged desserts), increase intracellular stress and lipid accumulation in immune cells. This metabolic burden promotes the activation of inflammasome complexes and perpetuates inflammation. Refined carbohydrates (white rice, white bread, crackers, pasta) lack fiber and antioxidants, further impairing glucose control and increasing inflammatory risk.

Western dietary pattern: The collective influence of a Western-style diet, which is high in saturated fats, added sugars, red and processed meats, and low in fiber, fruits, and vegetables, sets the stage for a pro-inflammatory internal environment. This diet has been shown to upregulate gene expression of inflammasome-related proteins and exacerbate systemic and cutaneous inflammation. In contrast, populations consuming traditional or plant-forward diets demonstrate lower rates of inflammatory skin disease, underscoring the role of dietary pattern in immune regulation [44,45].

Overall, consistent consumption of these pro-inflammatory dietary components may contribute to inflammasome hyperactivation and worsening of chronic dermatoses. Identifying and minimizing these triggers offers a valuable entry point for dietary interventions aimed at reducing skin inflammation and improving therapeutic outcomes.

Anti-inflammatory modulators: Numerous dietary components have demonstrated the ability to suppress NLRP3 inflammasome activation and downstream inflammatory signaling, offering promising adjunctive strategies in the management of inflammatory skin diseases. Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), long-chain omega-3 polyunsaturated fatty acids primarily found in fatty fish, have been shown to dampen NLRP3 activation through multiple pathways. By incorporating into cellular membranes, they improve membrane fluidity and reduce lipid raft formation, thereby modulating receptor signaling and immune cell activation [46]. Omega-3 fatty acids also reduce the production of pro-inflammatory eicosanoids and suppress NF-κB activation, leading to decreased transcription of NLRP3, IL-1β, and IL-18 [47]. In vitro and animal studies support their ability to attenuate skin inflammation, particularly in models of psoriasis and atopic dermatitis. Polyphenolic compounds found in plant-based foods, including resveratrol (grapes), curcumin (turmeric), and epigallocatechin gallate (EGCG; green tea), have emerged as potent inhibitors of inflammasome activation. These compounds exert anti-inflammatory effects by suppressing NF-κB signaling, reducing mitochondrial ROS production, and directly inhibiting NLRP3 assembly [48,49]. Vitamin D, through its active metabolite calcitriol (1,25-dihydroxyvitamin D3), modulates innate immunity by influencing the expression of NLRP3 and its downstream targets. Vitamin D receptor (VDR) activation has been shown to suppress NLRP3 transcription, inhibit caspase-1 activation, and reduce secretion of IL-1β in macrophages and dendritic cells [15]. Clinical studies suggest a correlation between low serum vitamin D levels and increased severity of psoriasis and atopic dermatitis, further supporting its immunomodulatory role in the skin [50]. Ketogenic diets, which are high in fat and low in carbohydrates, along with fasting-mimicking diets, have garnered interest for their systemic anti-inflammatory effects, in part due to their ability to suppress NLRP3 inflammasome activation. A key mediator of this effect is the ketone body β-hydroxybutyrate (BHB), which has been shown to directly inhibit NLRP3 activation. Rather than acting through a single pathway, BHB appears to modulate multiple cellular processes, including intracellular signaling, oxidative stress, endoplasmic reticulum stress, post-translational modifications, receptor activity, autophagy, and mitochondrial metabolism. These mechanisms likely vary depending on cell type, disease state, and timing, highlighting the potential of these dietary strategies in managing chronic inflammatory conditions [51].

Mechanisms of dietary modulation by signaling pathway

Dietary modulation impacts NLRPs in many ways and is the basis for certain inflammasome mechanisms in inflammatory dermatoses (Figure 1).

Autophagy/AMPK pathway: AMP-activated protein kinase (AMPK) is a serine/threonine kinase enzyme present in cells and conserves cellular energy. In a state of low energy, the protein kinase becomes activated due to the difference in the AMP: ATP ratio in cells. When the AMP level is higher, signaling cascades begin in cells to conserve energy by decreasing anabolic actions that use up energy and stimulating catabolic actions to preserve energy. Glucose uptake into cells is also stimulated by AMPK to provide energy to the cells [52]. In a

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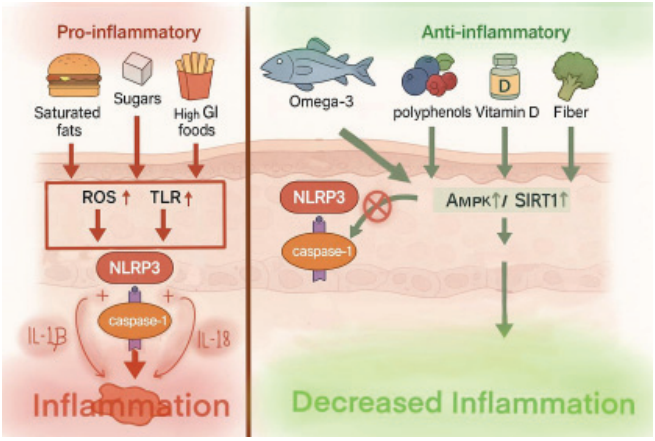

Figure 1: Molecular Pathways Linking Dietary Modulation to NLRP3 Inflammasome Activation.

state of low intracellular energy, liver kinase B1 (LKB1) a protein kinase which maintains cell energy homeostasis through activation of AMPK starts the cascade once AMPK detects the high AMP level, the conformation of AMPK changes which allows LKB1 to phosphorylates it to restore energy by limiting gluconeogenesis, glycogenesis, and fatty acid synthesis and increasing glucose and fat breakdown for energy usage [53,54]. AMPK signaling also influences NLRP3 inflammasomes during cellular energy crises. During the low energy state, AMPK initiates autophagy by Unc-51-like kinase 1 (ULK1), which initiates the clearance of NLRP3 inflammasome activators such as damaged cellular components, debris, cytokines, misfolded proteins, and DAMP [55,56]. AMPK signaling also inhibits nuclear factor kappa B (NF-kB), which decreases its transcription ability, decreasing inflammation in turn. NLRP3 activation is decreased [57,58]. Given the influences AMPK has, dietary modulation can influence NLRP3 inflammasomes. Western diets, which are high in saturated fat when consumed are metabolized into diacylglycerol (DAG) and ceramides, which are lipid intermediates. DAG, in turn, induces activation of protein kinase C (PKC), which phosphorylates and reduces AMPK's activity. Ceramides activate protein phosphatase 2A (PP2A), which also reduces AMPK's activity through dephosphorylation of AMPK [59]. These reductions in AMPK take away from the positive effect it has against NLRP3 and increase the risk of inflammasomes.

SIRT1/Nrf2 pathway: Silent information regulator sirtuin 1 (SIRT1) is a protein that has a regulatory mechanism through its NAD+ dependent deacetylase property on target proteins. Through acetylation, SIRT1 can influence inflammation indirectly through its signaling. This causes deacetylation of NF-kB, which limits inflammation. SIRT1 also enhances nuclear factor like 2 (Nrf2), a transcription factor with antioxidants that detoxifies mitochondrial ROS through enzymes, which in turn decreases NLRP3 inflammasome [60]. Foods high in fat cleave SIRT1 through caspase-1 triggered by inflammation due to excess lipids build up, taking away its anti-inflammasome activity [61,62].

Gut microbiome/butyrate pathway: Gut microbiome-mediated production of butyrate, a short-chain fatty acid (SCFA), provides energy for cells in the colon and regulates immunity [63]. Butyrate is formed through the fermentation of fibers and non-digestible carbohydrates. At optimal amounts, butyrate provides beneficial support to the colon along with inhibition of NF-kB, which limits cytokines and chemokines. Butyrate poses histone deacetylase inhibition (HDACi), which can decrease NF-kb, acting as a major anti-inflammatory and inhibiting genes that are pro-inflammatory [64]. Low presence of butyrate would decrease the ability of HDACi mechanism in decreasing expression of genes, as the chromatin will be tightly compacted together, and anti-inflammatory genes such as IL-10 would be suppressed, leaving NLRP3 inflammasome action unregulated [26]. Excessive saturated fatty acid (SFA) food can decrease gut microbiota that produce butyrate [65]. Mitochondrial ROS are introduced into cells as byproducts of the electron transport chain (ETC) reaction from electron leakage. The electron can then react with oxygen to form ROS, which makes the cellular environment harmful [66].

Reactive Oxygen Species (ROS) and antioxidant pathways: Build-up of ROS causes mitochondrial injury, which can result in mitochondrial DNA release (mtDNA) into the cell's cytoplasm. Apoptotic signals and NLRP3 will be activated through the caspase cascade, the mtDNA release also presents DAMP, which in turn also activates NLRP3 inflammasomes action [67,68]. Given the importance of mitochondrial ROS in influencing NLRP3, dietary modulation can play a major role. High-fat diet (HFD) can increase free fatty acids, which can be taken up by different cells. Inside the cell, they can enter the mitochondria to undergo beta-oxidation, but at high amounts, electron leakage from the mitochondria can occur and contribute to building up [69]. A healthy diet that consists of flavonoids, which scavenge ROS, and Omega-3 fatty foods, which have antioxidant properties are great alternative in maintaining mitochondrial optimal level to not create ROS and deactivate NLRP3 action [70,71].

Clinical implications for dermatology

Diet impacts the course of NLRP3, and it can be used as a clinical implication for dermatology to mitigate dermatomes (Table 2). Diet is not thought of first when assessing patients, and given the variety of ways the western diet impacts health, understanding NLRP3 implication from diets can enhance treatment options [72]. This can provide the patient with an additional tool that does not require more medication to fight the issue. In addition to biological or topical therapy, dietary modification of NLRP3 can provide an additional aid, as conditions such as psoriasis are associated with a high-fat, unbalanced diet along with the chronic inflammatory disease [73]. By using treatment along with diet modulation that induces chronic inflammation, there can be a positive impact at play. Though this provides a promising treatment, studies looking at diet and treatment together on NLRP3 are limited.

Personalized nutrition offers a promising and underutilized tool in the management of chronic inflammatory skin diseases. By integrating individualized dietary strategies into dermatologic care, clinicians and registered dietitians can address systemic inflammation at its nutritional root. This

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Table 2: NLRP3-Linked Inflammatory Skin Diseases and Dietary Interventions.

Skin Disease	NLRP3 Role / Evidence	Key Pro-Inflammatory Dietary Triggers	Effective Dietary Modulators	Suggested Nutrition Strategy
Psoriasis	↑ NLRP3, IL-1β, and caspase-1 in lesions	Saturated fats, sugars, Western diet	Omega-3s, polyphenols, vitamin D	Mediterranean or anti-inflammatory diet
Acne	↑ NLRP3 activation via P. acnes	High GI foods, dairy, saturated fats	Polyphenols, omega-3s	Low-GI, plant-based diet
Hidradenitis Suppurativa	↑ NLRP3 in lesional skin	Western diet, high fats, processed foods	Omega-3s, ketogenic diet	Calorie control, plant-forward, fasting
Atopic Dermatitis	↑ NLRP3 drives barrier dysfunction	Sugary snacks, trans fats, low fiber	Vitamin D, omega-3s, butyrate	High fiber, vitamin D-rich, low sugar

Abbreviations: NLRP3: NOD-like receptor protein 3; IL-1β: Interleukin-1 beta; IL-18: Interleukin-18; GI: Glycemic Index.

approach involves assessing a patient's unique inflammatory triggers, symptom patterns, skin condition subtype, lifestyle, and food environment to tailor dietary guidance that is both evidence-based and sustainable. Registered dietitians are uniquely positioned to translate complex scientific findings into clear, actionable dietary recommendations. Through detailed dietary recalls, food frequency questionnaires, and lifestyle assessments, Registered Dieticians (RDs) can identify pro-inflammatory dietary patterns (such as high saturated fat, trans fat, and refined sugar intake) and replace them with nutrient-dense, anti-inflammatory alternatives that align with the patient's cultural and practical preferences. For example, a patient with atopic dermatitis may benefit from increasing omega-3 fatty acid intake while reducing processed snacks and sugary beverages. Another with hidradenitis suppurativa might respond well to curcumin supplementation or a Mediterranean-style eating pattern that emphasizes polyphenol-rich produce and olive oil. Importantly, personalized nutrition must account for individual variability in genetics (e.g., nutrigenomics), gut microbiota composition, comorbid conditions, allergies, access to food, and behavioral readiness for change. Dietitians can provide meal planning support, culturally relevant food swaps, and education on reading food labels to reduce hidden pro-inflammatory ingredients. Incorporating food-based guidance into dermatology practice allows for a more holistic, systems-level approach that not only improves skin symptoms but also supports overall metabolic health, mood, and quality of life. Nutrition counseling can be adapted to personal preferences, cultural food practices, and coexisting medical conditions, ultimately enhancing patient adherence and improving long-term skin outcomes. As research continues to uncover the molecular links between diet and the skin immune system, especially through inflammasome pathways, registered dietitians will play a pivotal role in bridging the gap between nutritional science and dermatologic care.

Limitations & future directions

Despite significant advancements in understanding dietary modulation of the NLRP3 inflammasome in inflammatory skin diseases, several limitations persist. Most available studies examining dietary influence on NLRP3 activation are based on *in vitro* or animal models, which limits their applicability to human populations. Additionally, although NLRP3 is a well-established therapeutic target, no approved drugs currently target its pathway [74]. Future studies should aim to conduct large-scale clinical trials to assess long-term outcomes of dietary modifications in human subjects with various inflammatory skin diseases, which would potentially pave the way for the development of NLRP3-targeted therapies.

Another key limitation involves the need for reliable and validated biomarkers to monitor inflammasome activation and suppression. Currently, biomarkers such as IL-1β, IL-18, and ROS are used. However, these markers may not fully capture the full picture of inflammasome activity in vivo. The development and validation of gold-standard inflammasome-specific biomarkers are crucial for accurately monitoring treatment efficacy. Additionally, understanding genetic differences in inflammatory responses and nutrient metabolism could strongly guide personalized therapy. Future research should aim to incorporate nutrigenomic analysis, including the identification of relevant genetic markers and polymorphisms, to better predict patient responsiveness or resistance to the effect of dietary changes on NLRP3 activity.

Conclusion

Emerging research underscores the critical role of diet in modulating inflammasome activity, particularly the NLRP3 inflammasome, which serves as a central node in the pathophysiology of many chronic inflammatory skin diseases. Pro-inflammatory dietary components – such as saturated fats, high glycemic foods, and refined carbohydrates – can prime and activate the NLRP3 inflammasome through oxidative stress, mitochondrial dysfunction, and cytokine dysregulation. Conversely, anti-inflammatory nutrients and patterns, omega-3 fatty acids, polyphenols, vitamin D, and ketogenic or fasting-mimicking diets, demonstrate promising NLRP3-suppressive effects via mechanisms involving AMPK activation, SIRT1 signaling, autophagy induction, and modulation of gut microbiota-derived metabolites like butyrate. These findings hold significant clinical relevance for dermatology. Dietary counseling, particularly when individualized and evidence-based, offers a low-risk adjunct to standard treatments for conditions such as psoriasis, acne, atopic dermatitis, and hidradenitis suppurativa. Incorporating registered dietitians into dermatologic care teams and developing personalized nutrition strategies may enhance treatment efficacy, reduce flare frequency, and improve patients' quality of life. However, the field still faces notable limitations, including a paucity of dermatology-specific human trials, the need for validated biomarkers of inflammasome suppression, and a better understanding of nutrigenomic variability. As the interface between nutrition, immunity, and dermatology continues to evolve, future research must prioritize translational

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studies that bridge molecular insights with practical, scalable interventions. By recognizing the skin as both an immune and metabolic organ, clinicians can begin to leverage diet not just as background lifestyle advice, but as a targeted therapeutic modality for inflammatory dermatoses.

Conflict of interest

The authors declare that they have no economic or financial conflicts of interest related to this work. No honoraria, grants, consultancies, stock ownership, or other forms of compensation from organizations or entities with an interest in the subject matter of this manuscript were received. All authors have no conflicts to disclose.

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