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INFLUENCE OF THE GUT MICROBIOME ON THE PATHOGENESIS AND CLINICAL COURSE OF ATOPIC DERMATITIS

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ABSTRACT

Background: Atopic Dermatitis is a chronic inflammatory skin disease with a multifactorial pathogenesis involving genetic, immunological, and environmental factors. Recent research has highlighted the important role of the gut microbiome in modulating systemic immune responses and influencing the course of inflammatory diseases through the gut-skin axis.

Aim: The aim of this study was to review current knowledge on the impact of the gut microbiome on the pathogenesis and clinical course of atopic dermatitis.

Material and methods: A narrative literature review was conducted using databases such as PubMed, Scopus, and Web of Science. Articles from 2018-2026 were analyzed using keywords including „atopic dermatitis”, „gut microbiome”, „gut-skin axis”, „dysbiosis” and „probiotics”. The review included original studies, systematic reviews, and meta-analyses on the link between gut microbiota and atopic dermatitis.

Results: Alterations in gut microbiota composition (dysbiosis), including reduced microbial diversity and decreased abundance of beneficial bacteria, were associated with the development and severity of atopic dermatitis. The gut microbiome influences disease progression through immune system modulation, production of short-chain fatty acids, and regulation of intestinal barrier integrity. Evidence also suggests that microbiome-targeted therapies, such as probiotics and dietary interventions, may have beneficial effects on disease outcomes.

Conclusion: The gut microbiome significantly influences the development and course of atopic dermatitis. Understanding the gut-skin axis may help develop new therapies to restore microbial balance and improve outcomes.

KEYWORDS

Atopic Dermatitis, Gut Microbiome, Gut-Skin Axis, Dysbiosis, Probiotics, Immune System

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1. Introduction

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disorder characterized by intense pruritus, xerosis and skin redness [1]. It represents one of the most common dermatological diseases worldwide and significantly impairs quality of life [2]. The pathogenesis of AD is complex and multifactorial, involving genetic predisposition, immune dysregulation, environmental factors and impairment of the epidermal barrier [3]. In recent years, increasing attention has been given to the role of microbiota in AD, particularly the interaction between gut microbiome and skin health [4].

The global prevalence of AD has increased substantially over the past decades, although rates appear to be stabilizing in high-income countries. It affects up to 20% of children and 10% of adults, making it a major public health concern [5]. AD is marked by recurrent flare-ups and intermittent periods of remission. The disease usually follows a chronic, relapsing course, and its clinical manifestations vary according to the patient's age [6]. AD typically begins in infancy or early childhood and may constitute the first stage of the „atopic march”, preceding conditions such as asthma, rhinoconjunctivitis and eosinophilic esophagitis. Although it often resolves during childhood, the disease can persist into later life. In most adolescents and adults, AD represents a continuation of earlier disease, though it may also develop de novo during adolescence or adulthood [7]. A distinct subgroup of AD with onset at ≥ 60 years of age has recently been identified [8].

The concept of the gut-skin axis has emerged as a key framework for understanding the relationship between intestinal microbiota and dermatological conditions. The gut microbiome consists of trillions of microorganisms, including bacteria, viruses, fungi and archaea, that play essential roles in immune system development, metabolic processes and maintenance of host homeostasis [9]. Alterations in gut microbiota composition, known as dysbiosis, have been associated with various inflammatory and allergic diseases, including AD [10].

Evidence suggests that gut microbiota can influence skin inflammation through multiple mechanisms, including modulation of systemic immune responses, production of microbial metabolites such as short-chain

fatty acids (SCFAs) and regulation of epithelial barrier integrity. In patients with AD, dysbiosis is often characterized by reduced microbial diversity and altered abundance of specific bacterial taxa, which may contribute to disease onset and exacerbation [11].

Furthermore, the interplay between gut microbiota and the immune system is particularly relevant in AD, as the disease is associated with an imbalance of Th1 and Th2 immune responses, with predominance of the Th2 pathway in the acute phase of the disease. These cells contribute to the inflammatory process through the production of pro-inflammatory cytokines, including IL-4, IL-5 and IL-13, which are key mediators in the development and exacerbation of the disease [12]. Increased activity of these cytokines promotes IgE production, eosinophil activation, and chronic skin inflammation. Microbial signals from the gut can influence the balance of immune pathways, thereby affecting the severity and progression of skin inflammation. These findings highlight the potential of microbiome-targeted therapies in the management of AD [13].

The aim of this review is to summarize current knowledge regarding the role of the gut microbiome in the pathogenesis and clinical course of AD. Particular emphasis is placed on mechanisms linking gut dysbiosis to immune dysfunction, as well as potential therapeutic strategies targeting the gut microbiota.

2. Materials and Methods

2.1. Literature Search Strategy

A comprehensive literature search was conducted using electronic databases including PubMed, Scopus and Web of Science. The search covered publications from 2018 to 2026 to ensure inclusion of the most recent and relevant studies.

The following keywords and combinations were used: „atopic dermatitis”, „gut microbiome”, „gut-skin axis”, „dysbiosis”, „probiotics”, „microbiota”, „short-chain fatty acids”. Operators (AND, OR) were applied to refine the search.

2.2. Inclusion and Exclusion Criteria

Inclusion criteria:

- peer-reviewed articles published between 2018 and 2026
- original research studies, systematic reviews and meta-analyses
- studies focusing on gut microbiome in relation to AD

Exclusion criteria:

- articles not available in English
- case reports and conference abstracts
- studies unrelated to gut microbiome or AD

2.3. Data Extraction and Analysis

Relevant data were extracted and categorized based on study objectives, methodology and key findings. A qualitative synthesis approach was used to analyze the data, focusing on identifying common patterns and mechanisms linking gut microbiome alterations with AD pathogenesis.

3. Results

3.1. Composition and Function of the Gut Microbiome

The human body contains far more microbial cells than human cells, with gut bacteria being the most significant and prominent component of this microbiota ecosystem. The composition and abundance of these bacteria are influenced by various physiological factors, including intestinal motility, pH, redox potential, diet and host secretions such as gastric acid, bile, digestive enzymes and mucus. Additionally, the presence of an intact ileocecal valve plays an important role in shaping microbial distribution across different sections of the gut.

Beyond these intrinsic factors, external influences also contribute to variations in the gut microbiota. These include antibiotic use, disease, stress, aging, poor dietary habits and overall lifestyle.

Overall, the gut microbiome constitutes a highly complex and dynamic ecosystem, composed mainly of bacteria belonging to the phyla Firmicutes, Bacteroidetes, Proteobacteria, Actinobacteria, Euryarchaeota and Verrucomicrobia [14].

Microorganisms in the gastrointestinal (GI) tract play a crucial role in digestion, nutrient absorption, energy extraction, vitamin production, regulation of inflammation and maintenance of the host's immune system. The gut microbiota also competes with pathogenic microbes for nutrients and space, helping to prevent their colonization and potential invasion. In addition, it produces various bioactive compounds, including SCFAs, which have anti-inflammatory properties and support the integrity of the gut barrier [15].

The gut microbiota regulates immune function both systemically and cutaneous immune responses. It influences the differentiation of naïve T cells into Th1, Th2, Th17 or Foxp3 Treg cells, which help control inflammation by suppressing overactive immune cells and regulating antibody production. Beneficial bacteria (Bifidobacterium, Lactobacillus, Clostridium, Bacteroides and Streptococcus) and their metabolites, such as propionate and butyrate, are known to promote Treg cell polarization and expansion [16].

Table 1. Factors Influencing Gut Microbiome Composition [14]

Category	Factors	Impact on the Gut Microbiome
Endogenous factors	Intestinal motility	Regulates the contact time between bacteria and intestinal contents
Endogenous factors	Intestinal pH	Selects for specific bacterial groups
Endogenous factors	Redox potential	Influences microbial growth conditions
Endogenous factors	Gastric acid, bile, digestive enzymes	Limits colonization by pathogens
Endogenous factors	Intestinal mucus	Creates a barrier and a niche for microbiota
Endogenous factors	Intact ileocecal valve	Limits bacterial migration between the small and large intestine
Exogenous factors	Antibiotics	Reduces microbial diversity
Exogenous factors	Diet	Alters bacterial composition (e.g., SCFA production)
Exogenous factors	Stress	Disrupts the gut–brain axis
Exogenous factors	Aging	Decreases microbial diversity
Exogenous factors	Lifestyle	Overall modulation of the microbiome

3.2. Gut Dysbiosis in Atopic Dermatitis

Each individual develops a unique composition of gut microbiota. Once established, this microbial community tends to remain relatively stable throughout life. Dysbiosis is defined as a state characterized by the loss of beneficial microorganisms, an overgrowth of potentially harmful species or a reduction in overall microbial diversity [17].

Studies have demonstrated alterations in the gut microbiome of patients with AD compared to healthy individuals. These changes include an increased abundance of *Clostridium difficile* and a reduced presence of SCFA-producing bacteria, such as *Bifidobacterium*. SCFAs are essential for maintaining host health and their decreased production may contribute to intestinal inflammation in individuals with AD [18].

Decreased exposure to SCFAs during early life may contribute to the development and progression of AD. For example, Roduit et al. found that lower fecal levels of propionate and butyrate in one-year-old children were associated with a higher risk of atopic diseases by age six. Similar findings indicate that reduced butyrate levels in infancy are linked to the development of eczema, while lower exposure to valerate may also be associated with increased eczema risk [19].

Infants with AD exhibit reduced gut microbiota diversity compared to healthy controls. This is characterized by higher levels of potentially pro-inflammatory bacteria, such as *Clostridium difficile*, *E. coli* and *Bacteroides*, alongside decreased abundance of beneficial microbes, including *Akkermansia*, *Lactobacillus*, *Faecalibacterium prausnitzii* and *Bifidobacterium* [20].

The gut microbiome also differs markedly between AD adult patients and healthy individuals. A study by Wang et al. showed that beneficial genera such as *Romboutsia* and *Clostridium sensu stricto* 1 were more abundant in healthy controls, while *Blautia*, *Butyrivibrio*, *Lachnospirillum*, *Eubacterium hallii* group, *Erysipelatoclostridium*, *Megasphaera*, *Oscillibacter* and *Flavonifractor* predominated in AD patients [21].

Table 2. Gut microbiome alterations in Atopic Dermatitis [17-21]

Feature	Healthy individuals	Patients with AD
Microbial diversity	High	Reduced
Bifidobacterium	High	Decreased
Lactobacillus	High	Decreased
Pro-inflammatory bacteria	Low	Increased
SCFA levels	Normal	Reduced

3.3. Mechanisms Linking Gut Microbiome and Atopic Dermatitis

3.3.1. Immune System Modulation

The gut microbiota plays a crucial role in regulating both systemic and skin immunity. It significantly influences the differentiation of naïve T cells into Th1, Th2, Th17 or Foxp3 Treg cells. Tregs help prevent excessive T cell activation and suppress the inflammatory responses of immune cells such as mast cells, eosinophils and basophils. Additionally, Th cells modulate antibody production by inhibiting IgE and promoting IgG4 synthesis. Commensal bacteria stimulate the development of regulatory T cells, which are essential for maintaining immune tolerance and preventing excessive inflammatory responses [16].

In the context of AD, immune dysregulation is characterized primarily by a predominance of T helper 2 (Th2)-mediated responses, leading to increased production of interleukins such as IL-4, IL-5 and IL-13 which ultimately lead to increased production of IgE, commonly observed in patients with AD [13].

3.3.2. Microbial Metabolites and Short-Chain Fatty Acids (SCFAs)

Short-chain fatty acids (SCFAs), primarily acetate, propionate, butyrate, isobutyrate, valerate and isovalerate, are key metabolites produced by bacterial fermentation of dietary fibers in the gut. SCFAs produced by gut microbiota, play an important role in modulating the host inflammatory response and maintaining physiological homeostasis. Increasing evidence suggests that AD may arise, at least in part, from systemic immune disturbances driven by alterations in gut microbial composition. Notably, reduced intestinal levels of SCFAs have been associated with the onset of AD. Patients with AD typically exhibit lower fecal concentrations of SCFAs, reflecting a diminished presence of SCFA-producing bacteria. This reduction may compromise intestinal barrier integrity, enhance gut permeability and thereby contribute to the development and progression of AD [22].

In particular, gut microbiota imbalance in AD is associated with disrupted SCFA metabolism, especially decreased butyrate levels. Butyrate exerts strong anti-inflammatory effects by interacting with intestinal epithelial cells and immune cells, including macrophages, B cells and T cells, while also regulating T cell differentiation. In children with AD, significant disturbances in SCFA metabolism have been observed, including reduced levels of butyrate and a decline in butyrate-producing bacteria with age, which negatively correlates with serum IgE levels. Additionally, the downregulation of key genes involved in butyrate synthesis and signaling, such as GPR109A and PPAR- γ , further highlights the role of impaired SCFA pathways in the pathogenesis of AD, particularly in older infants [23].

3.3.3. Intestinal Barrier Dysfunction

The integrity of the intestinal barrier is essential for preventing systemic exposure to harmful microbial products. Disruption of this barrier, often referred to as „leaky gut” has been implicated in the pathogenesis of multiple inflammatory conditions, including AD. Increased intestinal permeability allows microbial antigens and toxins to enter the bloodstream, triggering systemic immune responses and contributing to inflammation. Immune cells activated in the gut can migrate to the skin, where they contribute to the development of AD. The skin immune response is closely linked to the cytokine profile generated in the gut, as reflected by elevated levels of circulating inflammatory cytokines [24].

Butyrate-producing gut bacteria, including Bifidobacterium, Lactobacillus, Clostridium, Bacteroides, and Streptococcus, along with their metabolites, play an important role in regulating immune responses. They support the proliferation and activation of regulatory T (Treg) cells and help reduce intestinal epithelial permeability, thereby influencing inflammatory processes in extraintestinal organs such as the skin. Moreover, Nyland et al. demonstrated that a lower abundance of these microbes is associated with greater disease severity in infants with AD [25].

In addition to the gut microbiome, diet plays a significant role in the development and progression of AD. Dietary factors can directly influence gut barrier integrity and microbial composition. A gluten-containing

diet may impair intestinal barrier function. This disruption facilitates the translocation of microbial products and antigens into the bloodstream, which can trigger systemic immune responses. Moreover, gluten sensitivity has been associated with alterations in gut microbiota composition, further contributing to dysbiosis and potentially exacerbating inflammatory conditions such as AD [13].

3.4. Influence of Gut Microbiome on Disease Severity and Progression

The gut microbiome plays a significant role in the severity and course of AD. Patients with more severe forms of the disease often show reduced levels of beneficial bacteria such as Bifidobacterium and Bacteroidetes, which help regulate immune responses and limit inflammation. The presence of other groups of microorganisms is also of significant importance. For example, the number of Streptococcus positively correlates with the SCORAD index, which is used to assess the severity of AD, whereas the presence of bacteria from the Clostridium genus shows an inverse correlation. Differences in microbiome composition may also influence whether the disease follows a transient or chronic course [26].

Several studies demonstrated significant correlations between gut microbiota composition and AD severity assessed using the SCORAD index. Liu et al. reported that infants with severe AD had lower levels of Clostridium sensu stricto and increased abundance of Parabacteroides and Bacteroides. Importantly, the relative abundance of Bacteroides positively correlated with SCORAD scores, whereas Clostridium sensu stricto showed an inverse correlation, suggesting a protective role of butyrate-producing bacteria in AD progression [27].

Dietary habits and environmental factors substantially influence microbiome composition and may affect AD severity. Diets high in sugar and saturated fats can reduce microbial diversity and decrease SCFA production, thereby promoting intestinal permeability and systemic inflammation. Similarly, frequent antibiotic exposure may disrupt gut microbial balance by eliminating commensal bacteria and facilitating colonization by potentially pathogenic microorganisms. These changes may aggravate AD symptoms and contribute to recurrent disease exacerbations [26].

3.5. Therapeutic Strategies Targeting the Gut Microbiome

3.5.1. Probiotics

Probiotics are live microorganisms that, when consumed in adequate amounts, confer beneficial effects on the host's health. Beyond modulating the gut microbiota, they exhibit significant effects in experimental and preclinical studies. In particular, strains of Lactobacillus and Bifidobacterium are well known for their beneficial properties. Probiotics also exert immunomodulatory effects and produce bioactive compounds, such as enzymes, SCFAs and vitamins, which contribute to overall host health [24].

Probiotic supplementation is becoming increasingly popular, especially in children, as a strategy to modulate the gut microbiota and regulate immune function. Meta-analyses suggest that certain probiotic strains can reduce the severity of AD, as reflected by improvements in SCORAD scores in adults. Additionally, Fang et al. demonstrated that specific strains, such as Bifidobacterium CCFM16 and Lactobacillus plantarum CCFM8610, may alleviate AD symptoms by modifying the composition and activity of the gut microbiota [28].

Probiotics play an important role in shaping the immune system, particularly in early life, supporting the concept of the hygiene hypothesis. They contribute to the development of immune tolerance and help maintain intestinal immune homeostasis, for example by stimulating IgA production, which enhances mucosal defense and protects against pathogen adhesion. Specific strains such as Bifidobacterium and Lactobacillus have been shown to modulate immune responses by promoting a balance between Th1 and Th2 cells, enhancing regulatory T cell differentiation and suppressing excessive Th2-driven inflammation associated with allergic diseases like AD. Additionally, probiotics can reduce the production of pro-inflammatory cytokines, including IL-4, IL-5, IL-13 and TSLP, which are key mediators in the pathogenesis of AD. Through these mechanisms, probiotics help alleviate inflammation, improve skin barrier function and reduce disease severity, highlighting their potential as a supportive therapeutic strategy in AD management [29].

3.5.2. Prebiotics and Diet

Diet plays a crucial role in shaping the gut microbiota by providing substrates that support the growth of specific microbial populations. In particular, dietary fiber (composed of non-digestible polysaccharides) serves as a key substrate for microbial fermentation. These compounds, known as prebiotics, selectively promote the growth of beneficial bacteria and contribute to host health. Well-established prebiotics include

inulin, fructo-oligosaccharides (FOS), galacto-oligosaccharides (GOS) and lactulose, while emerging candidates such as polyphenols, polyunsaturated fatty acids and human milk oligosaccharides are also being investigated [24].

Prebiotics stimulate the production of short-chain fatty acids such as acetate, propionate and butyrate, which exert anti-inflammatory effects. They also help limit the formation of harmful fermentation by-products, promote the proliferation of immune cells in gut-associated lymphoid tissue and enhance the secretion of intestinal IgA [26].

Prebiotics have shown beneficial effects in patients with AD, as clinical studies indicate that their use can alleviate disease symptoms and reduce levels of pro-inflammatory mediators, highlighting their potential as supportive therapeutic strategies in AD management [24]. Notably, one study demonstrated that supplementing infant diets with prebiotic oligosaccharide mixtures during the first year of life reduced the incidence of AD by approximately 44% [16].

In addition to prebiotics, fermented foods such as kefir may also provide benefits for patients with AD. Regular consumption of kefir has been shown to improve skin barrier function, as demonstrated by reduced transepidermal water loss (TEWL) and increased skin hydration in patients with AD. Moreover, kefir intake was associated with a significant reduction in disease severity, reflected by decreased SCORAD scores. These effects are likely mediated by kefir's rich probiotic composition, which can favorably modulate the gut microbiota, enhance immune regulation and reduce systemic inflammation [30].

Overall, both prebiotics and probiotic-rich foods like kefir appear to be promising dietary adjuncts in the management of AD, although further well-controlled human studies are needed to confirm their efficacy.

3.5.3. Synbiotics and Emerging Therapies

Synbiotics, defined as combinations of probiotics and prebiotics, can beneficially modulate the gut microbiota and support both intestinal and skin health. They promote the growth of beneficial bacteria, inhibit pathogenic microorganisms, enhance immune function (e.g., by increasing intestinal IgA levels) and may improve mineral absorption while reducing harmful metabolic by-products in the gut [26].

Importantly, synbiotics show promising effects in AD. They can reduce skin inflammation, modulate the immune response by lowering IgE and Th2-related cytokines, and increase the abundance of SCFA-producing bacteria. As a result, synbiotics may help alleviate AD symptoms and support systemic immune balance, highlighting their potential as a supportive dietary strategy [31].

Fecal microbiota transplantation (FMT) has recently emerged as a potential strategy for modulating the gut microbiome, particularly in conditions associated with dysbiosis. FMT involves the transfer of functional microorganisms from the feces of a healthy donor into the gastrointestinal tract of a recipient, with the aim of restoring microbial balance. Increasing evidence suggests that the gut microbiota may influence various skin conditions, including acne, psoriasis, hair loss and eczema, highlighting the relevance of the gut-skin axis [16].

In the context of AD, FMT is considered one of the most direct approaches to correcting gut dysbiosis. A study by Mashiah et al. reported significant improvements in clinical symptoms, assessed using the SCORAD index, following FMT in a small group of patients, without notable adverse effects. However, despite these promising findings, the use of FMT remains controversial due to the potential risk of serious complications and the lack of standardized treatment protocols. Consequently, further research is required to establish its safety and efficacy in AD management [25].

Table 3. Microbiome-targeted therapies in AD [24, 26, 31]

Therapy	Mechanism	Clinical outcome
Probiotics	Restoration of beneficial bacteria	Reduction of disease severity
Prebiotics	Stimulation of beneficial microbiota growth	Improved gut balance
Diet	Increased fiber intake → SCFA production	Anti-inflammatory effect
Synbiotics	Combined probiotic + prebiotic effect	Enhanced therapeutic outcomes

4. Discussion

The findings presented in this review highlight the complex and multidimensional relationship between the gut microbiome and AD, supporting the concept of the gut-skin axis as a key factor in the pathogenesis and progression of this disease. The gut microbiota, as a dynamic and metabolically active ecosystem, plays a central role not only in digestion and nutrient metabolism but also in immune regulation and maintenance of epithelial barrier integrity. Disruptions in this system, particularly in the form of dysbiosis, appear to contribute significantly to the development and severity of AD.

One of the most consistent observations across studies is the altered composition of the gut microbiome in patients with AD. Reduced microbial diversity and a decreased abundance of beneficial bacteria, such as *Bifidobacterium* and *Lactobacillus*, are frequently reported, alongside an increased presence of potentially pathogenic or pro-inflammatory species, including *Clostridium difficile* and *Escherichia coli*. These alterations may impair immune tolerance and promote chronic inflammation. Importantly, such changes are already evident in early life, suggesting that the initial establishment of the gut microbiota may have long-term consequences for immune development and susceptibility to allergic diseases.

A key mechanism linking gut dysbiosis to AD involves microbial metabolites, particularly SCFAs. SCFAs, such as acetate, propionate and butyrate, play a crucial role in maintaining intestinal homeostasis, regulating immune responses and preserving epithelial barrier function. Reduced levels of SCFAs, commonly observed in patients with AD, may lead to increased intestinal permeability, often referred to as „leaky gut“. This condition facilitates the translocation of microbial antigens and toxins into the systemic circulation, triggering immune activation and promoting inflammation in distant organs, including the skin. Moreover, butyrate has been shown to exert strong anti-inflammatory effects and to support the differentiation of regulatory T cells, which are essential for maintaining immune tolerance. Therefore, impaired SCFA production represents a critical link between gut microbiota alterations and immune dysregulation in AD.

Immune imbalance is another fundamental aspect of AD pathogenesis associated with the gut microbiome. The predominance of Th2-mediated immune responses, characterized by increased production of cytokines such as IL-4, IL-5 and IL-13, leads to elevated IgE levels and enhanced allergic inflammation. The gut microbiota influences the differentiation of naïve T cells into various subsets, including Th1, Th2, Th17 and Treg cells. A reduction in Treg activity, often associated with dysbiosis, may further exacerbate immune dysregulation. Additionally, immune cells activated in the gut can migrate to the skin, where they contribute to local inflammation, highlighting the systemic nature of the gut-skin axis.

The integrity of the intestinal barrier also plays a pivotal role in the interaction between the gut microbiome and AD. Disruption of this barrier allows increased passage of microbial components into the bloodstream, which can amplify systemic inflammation and worsen skin symptoms. Dietary factors, such as high sugar intake or gluten sensitivity, may further impair barrier function and contribute to dysbiosis. These findings emphasize the importance of environmental and lifestyle factors in modulating disease risk and progression.

Given the central role of the gut microbiome in AD, therapeutic strategies targeting its modulation have gained increasing attention. Probiotics, particularly strains of *Lactobacillus* and *Bifidobacterium*, have shown promising results in improving clinical outcomes in AD. Their beneficial effects are attributed to their ability to restore microbial balance, enhance SCFA production, regulate immune responses and strengthen the intestinal barrier. Meta-analyses indicate that certain probiotic interventions can reduce disease severity, as measured by SCORAD scores, although results remain somewhat inconsistent due to differences in study design, strains used and treatment duration.

Similarly, prebiotics and dietary interventions represent an important approach to modulating the gut microbiome. By promoting the growth of beneficial bacteria and increasing SCFA production, prebiotics may help reduce inflammation and improve immune function. Notably, early-life supplementation with prebiotic oligosaccharides has been associated with a reduced risk of developing AD, suggesting a potential preventive role. Fermented foods, such as kefir, also appear to exert beneficial effects, likely due to their combined probiotic content and bioactive metabolites, which support both gut and skin health.

Emerging therapies, including synbiotics and FMT, offer additional avenues for intervention. Synbiotics may provide synergistic effects by combining the benefits of probiotics and prebiotics, enhancing microbial balance and immune regulation. FMT, as a more direct method of restoring gut microbiota composition, has shown preliminary promise in small-scale studies, with improvements in AD symptoms reported. However, concerns regarding safety, standardization and long-term outcomes currently limit its clinical application.

Despite the growing body of evidence supporting the role of the gut microbiome in AD, several limitations must be acknowledged. Many studies are heterogeneous in design, involve small sample sizes or lack long-term follow-up. Additionally, the complexity of host–microbiome interactions makes it difficult to establish clear causal relationships. Future research should focus on large-scale, well-controlled clinical trials, as well as mechanistic studies aimed at elucidating the precise pathways involved.

In conclusion, the gut microbiome plays a critical role in the pathogenesis and progression of AD through its effects on immune regulation, metabolic activity and barrier integrity. Modulation of the gut microbiota represents a promising therapeutic strategy with probiotics, prebiotics and dietary interventions showing potential benefits. However, further research is needed to optimize these approaches and translate them into effective, evidence-based clinical practice.

5. Conclusions

The gut microbiome plays a crucial role in the pathogenesis and clinical course of AD. Dysbiosis, characterized by reduced microbial diversity and altered composition, contributes to immune dysregulation and inflammation.

Understanding the mechanisms linking gut microbiota and skin health opens new avenues for therapeutic interventions. Strategies targeting the gut microbiome, including probiotics, dietary modifications and emerging therapies, may offer promising approaches for improving disease outcomes.

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