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From Diet to Dermis: Understanding the Gut-Skin Axis- A Narrative Review

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Abstract

BACKGROUND: The gut–skin axis represents a bidirectional network linking intestinal microbiota, immune regulation, neuroendocrine signaling, and skin barrier function. The gut and skin share structural, immunologic, and neurovascular similarities, enabling systemic communication through microbial metabolites, cytokines, and hormones. Gut-derived SCFAs, vitamins, and immune mediators maintain epithelial integrity and modulate cutaneous inflammation, while dysbiosis and barrier disruption promote systemic inflammatory responses affecting skin homeostasis. Diet critically modulates gut microbial composition and metabolite production. Increasing evidence links gut–skin axis dysregulation to inflammatory and autoimmune skin disorders such as acne, psoriasis, AD, rosacea, and vitiligo.

AIM: To assess the role of the gut–skin axis and diet in skin health.

METHODS1: This is a narrative review. Literature was searched in PubMed, Web of Science, CNKI, and ScienceDirect for articles published between 2006 – 2025. Keywords included "gut–skin axis," "gut microbiota," "skin inflammation," "diet," "SCFAs," and "neuroendocrine signaling." Original research articles, reviews, and meta-analyses published in English were included. Case reports and non-peer-reviewed articles were excluded.

DISCUSSION: The gut–skin axis regulates skin health through microbial metabolites, immune balance, and diet. Dysbiosis-driven inflammation contributes to conditions like acne, psoriasis, and atopic dermatitis, highlighting the potential of microbiota- and nutrition-targeted interventions to improve dermatologic outcomes.

CONCLUSION: The gut–skin axis integrates microbiota, immune, dietary, and neuroendocrine pathways essential for skin homeostasis. Gut dysbiosis and barrier dysfunction contribute to inflammatory and autoimmune skin diseases. Targeting diet and microbiota modulation offers promising preventive and therapeutic strategies in dermatologic care.

KEYWORDS: Gut skin axis, Gut microbiota, Diet, Neuroendocrine signaling, Inflammatory skin diseases

Introduction

The link between the gut and the skin is widely acknowledged, as gastrointestinal disorders are frequently accompanied by skin manifestations[1]. Stokes and Pillsbury suggested that emotional states may disturb the normal balance of intestinal microbes, increase gut permeability, and trigger inflammation throughout the body[2]. Because the gut and skin serve important barrier functions at the body's interfaces, it is

unsurprising that various gastrointestinal disorders are associated with skin conditions[3]. Although gut microbiota are essential to host health, growing evidence suggests they may also contribute to the development of various diseases[4].

The gut–skin axis refers to bidirectional communication involving the gut microbiota, intestinal barrier, immune system, and skin. Research suggests this network supports skin homeostasis and may influence inflammatory skin conditions. Gut microbes generate compounds such as short-chain fatty acids and bile acids, which can affect immune activity and skin responses, highlighting the microbiome's importance to both skin health and gastrointestinal function[5]. The microbiota is crucial for the development, education, and function of the host immune system. In turn, the immune system has evolved primarily to maintain a mutually beneficial relationship between the host and its diverse, ever-changing microbial communities[6].

The skin is continually exposed to internal and external stressors, and psychological stress can affect its function and contribute to skin disorders[7]. As the body's main barrier, it contains immune cells that help regulate skin responses. In psoriasis, inflammatory dendritic cells activate T cells, which release cytokines that drive inflammation. The effectiveness of treatments targeting these cytokines underscores their role in the disease[8]. Stress-related hormones, including cortisol, ACTH, and CRH, can trigger skin immune responses, while skin cells may also produce these mediators and promote local inflammation[9]. Mind–skin communication involves interactions between the brain, immune system, and skin, and psychological processes may contribute to common skin disorders and lesion development[10].

Rationale for the Review:

The interplay between the gut, skin, and neuro-immuno-endocrine systems—collectively described as the gut–skin axis—has emerged as a critical factor in maintaining skin health and mediating inflammatory and stress-related dermatologic conditions. Although numerous studies have examined individual components, such as microbiota, immune signaling, or stress mediators, the integrated mechanisms linking gut dysbiosis, immune modulation, and psychoneuroendocrine influences on skin remain scattered and incompletely understood. This review aims to provide a comprehensive synthesis of current evidence on the gut–skin axis, elucidating the roles of microbial metabolites, immune responses, and neuroendocrine pathways in skin physiology and pathology, with implications for therapeutic interventions.

This review aims to synthesize current knowledge on the gut–skin axis, highlighting interactions between gut microbiota, immune responses, and neuroendocrine pathways in maintaining skin health and mediating inflammatory skin diseases. It examines the roles of microbial metabolites, cytokines, and stress mediators, and discusses potential therapeutic interventions targeting this axis. The scope is limited to mechanisms linking the gut and skin, excluding unrelated gastrointestinal or dermatologic conditions.

Gut–Skin Axis: Biological Basis

The gut and skin have many functional similarities and are both richly innervated and supplied with blood, reflecting their roles in immune and neuroendocrine activity[11]. The gut microbiota comprises microorganisms living in the gastrointestinal tract—including bacteria, archaea, fungi, and some protozoa and algae—along with their genes and metabolic products. The intestinal environment, shaped by substances from both the host and microbes, is also part of this ecosystem[12]. In the epidermis, keratinocytes and Langerhans cells are the primary cell types, with Langerhans cells acting as key antigen-presenting cells. The dermis contains a wider variety of immune cells, including dendritic cells and

macrophages, as well as sensory nerves involved in neuroimmune communication and cytokine signaling[13]. Gut-derived compounds can affect skin receptors or interact with skin-resident microbes. This communication is two-way: skin-related factors, such as UVB exposure, may also influence gut microbiota. For example, UVB exposure can increase serum vitamin D levels, potentially promoting greater microbial diversity in the gut[5]

Table 1. Key microbial metabolites and their effects on the gut-skin axis[14]

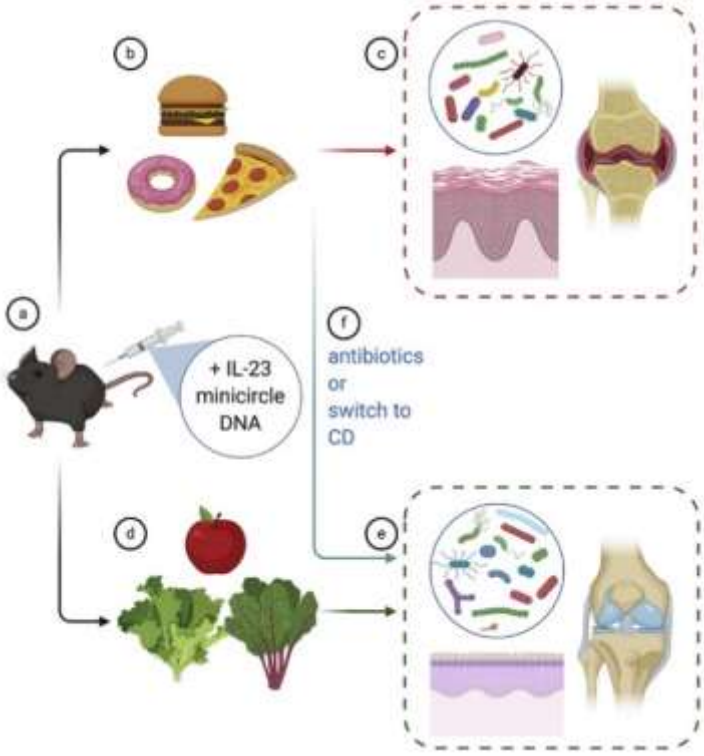
Metabolite	Produced by	Effects on gut	Effects on skin
SCFAs (*e.g.*, butyrate)	Firmicutes (*e.g.*, *Faecalibacterium*)	Enhances gut barrier, anti-inflammatory	Promotes tregs, reduces inflammation
p-Cresol	*Clostridium difficile*	Increases gut permeability	Decreases keratinocyte differentiation, causes xerosis
Indole derivatives	Tryptophan metabolism	Modulates intestinal immunity *via* AhR	Influences skin inflammation and barrier repair
Zonulin	Enteric epithelial cells	Increases intestinal permeability ("leaky gut")	May indirectly increase risk of inflammation-related dermatoses
Serotonin	Enterochromaffin cells/gut flora	Gut motility, mood regulation	Implicated in pruritus and neurogenic inflammation
Trimethylamine-N-oxide	Gut bacteria metabolizing choline/carnitine (*e.g.*, *Lachnoclostridium*)	Increases systemic inflammation and oxidative stress; implicated in metabolic dysfunction	May contribute to skin aging and inflammatory dermatoses (indirectly *via* systemic effects)

Disruption of the gut barrier and imbalance in the microbiota can undermine skin homeostasis through the gut–skin axis. Gut microbes help regulate inflammation throughout the body, including in the skin, by supporting intestinal barrier integrity and immune balance. Commensal bacteria produce vitamins and short-chain fatty acids, particularly butyrate, that strengthen epithelial tight junctions and reduce intestinal permeability. The mucus layer, antimicrobial peptides, secretory IgA, and innate immune cells in gut-associated lymphoid tissue also help stop microbes from crossing the intestinal barrier. Antigen presentation by dendritic cells promotes the development of regulatory T cells, IgA-producing B cells, and Th17 cells; Th17-derived IL-22 further supports antimicrobial defenses. Together, these mechanisms protect the gut barrier, limit systemic inflammation, and help preserve skin homeostasis[15]

Role of Diet in Modulating the Gut–Skin Axis

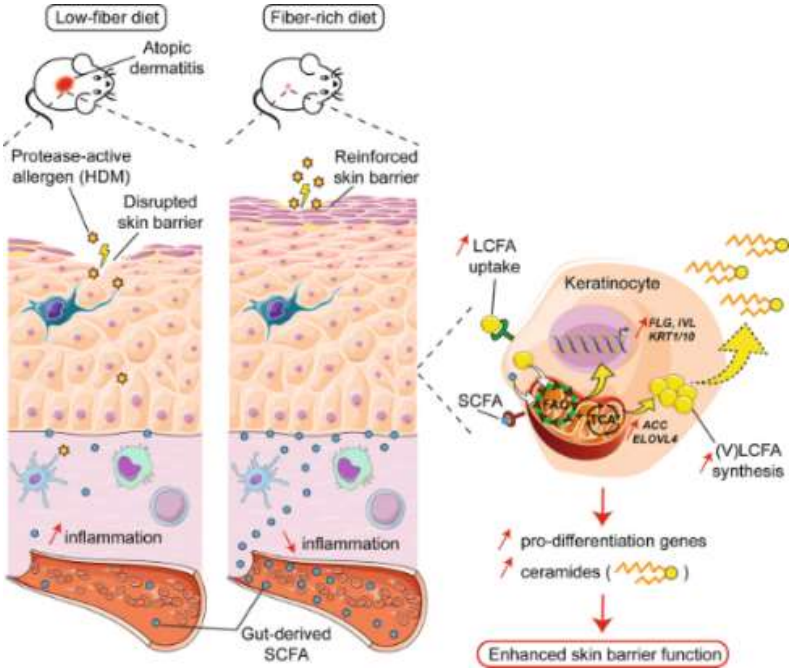
The Western diet is typically rich in calorie-dense but nutrient-poor foods, including fast food, soft drinks, and processed products containing added sugars, salt, and saturated fats. By comparison, traditional eating patterns such as the Mediterranean diet focus on fruits, vegetables, whole grains, legumes, and healthful fat[16]. Shi et al. (2021) reported that a Western diet (WD) aggravates IL-23-driven psoriasis and psoriatic arthritis in mice, increasing IL-17A-expressing γδ T cells and altering the gut microbiota. Dietary modification or antibiotic treatment reduced WD-associated inflammation, indicating that diet changes may help manage IL-23-mediated conditions by influencing the gut microbiome[17].

Fig.1 Western diet-induced IL-23 mediated skin and joint disease in mice can be alleviated by antibiotics or switching to a conventional diet[17]



Barrier dysfunction promotes allergic disease, including atopic dermatitis, and is associated with reduced dietary fiber–derived SCFAs. A fermentable fiber–rich diet mitigates AD-like inflammation via the gut–skin axis, primarily through butyrate-mediated enhancement of epidermal barrier integrity. This limits allergen sensitization and disease progression.

Fig 2. Gut-derived short-chain fatty acids modulate skin barrier integrity by promoting keratinocyte metabolism and differentiation (18)



Probiotics support the skin's defenses by promoting antimicrobial peptide production and stimulating keratinocyte differentiation and renewal[19]. Probiotic treatments have recently been proposed as a way to improve skin health, alongside the growing interest in microbiome-focused skin care. By influencing skin health and gut–skin axis interactions, probiotics may help manage skin disorders through several mechanisms, including reducing oxidative stress, limiting inflammation, and modulating immune activity[20]. In models of obesity induced by high-fat diets, omega-3 polyunsaturated fatty acids (PUFAs) improve gut health by helping restore microbial balance. They consistently lower the Firmicutes/Bacteroidetes ratio, increase beneficial bacteria such as *Akkermansia muciniphila*, *Bifidobacterium*, and *Lactobacillus*, boost short-chain fatty acid production, strengthen gut barrier function, and reduce pathogenic bacteria. These findings suggest that omega-3 PUFAs may protect the gut by shaping the microbiota, supporting their potential as a dietary strategy for improving gut-related metabolic and inflammatory outcomes[21].

Table 2. Selected Dietary and Microbiota-Targeted Interventions Relevant to the Gut–Skin Axis: Study Characteristics and Key Findings

AUTHOR AND YEAR	JOURNAL	STUDY MODEL	INTERVENTION	KEY QUANTITATIVE RESULTS
Smith RN et al. (2007)	Am J Clin Nutr. 2007 July;86(1):107–15.	Human RCT	Low-glycemic-load diet	Acne lesion reduction: −23.5 ± 3.9 vs −12.0 ± 3.5 (P=0.03); BMI ↓ −0.92 ± 0.25 (P=0.001); Insulin sensitivity improved (P=0.026)
Trompette A et al. (2022)	Mucosal Immunol. 2022 Aug;15(5):908–26.	Animal + translational	High-fiber diet / SCFA	Butyrate ↑ ~2-fold; skin inflammation ↓ 45–60%; epidermal barrier restored
Cao B et al. (2025)	Diabetes Obes Metab. 2025 Sept;27(9):4643–61.	Systematic review	Omega-3 PUFA supplementation	Akkermansia ↑ 2–4-fold; SCFA ↑ ~40%; gut permeability ↓
Abdi A et al. (2024)	Biochem Biophys Res Commun. 2024 Apr;702:149632.	Experimental + human	Probiotics	IL-6 & TNF-α ↓ 20–35%; oxidative stress ↓ ~25%; keratinocyte differentiation ↑ ~30%
Belkaid Y, Hand TW (2014)	Cell. 2014 Mar;157(1):121–41.	Mechanistic review	SCFA-immune modulation	NF-κB ↓ ~50%; Treg cells ↑ ~2-fold; systemic

				inflammation reduced

5. Diseases Associated with Gut–Skin Axis

5.1 Acne Vulgaris

Pathophysiology:

Acne results from pilosebaceous unit dysfunction with abnormal keratinization, *Cutibacterium acnes* colonization, sebum overproduction, and inflammation. Diet can influence hormonal and immune pathways that affect sebum and follicular inflammation[22]. The improvement in acne and insulin sensitivity after a low-glycemic-load diet suggests that nutrition-related lifestyle factors may play a role in the pathogenesis of acne[23]

Gut Microbiota Alterations:

Systematic reviews suggest that people with acne frequently have imbalances in their gut microbiota, including shifts in the Firmicutes-to-Bacteroidetes ratio and an increased abundance of Proteobacteria. These findings point to a possible connection between gut dysbiosis and systemic inflammation, which may intensify skin inflammation[1]

Dietary Influences:

Diets with a high glycemic index and dairy consumption are linked to more severe acne, potentially through increased IGF-1 and inflammatory signaling. In contrast, low-glycemic-index and plant-based diets rich in fiber and antioxidants are associated with reduced acne severity[22].

5.2 Psoriasis

Pathophysiology:

Psoriasis is a chronic, immune-mediated skin disease driven by Th17/Th1 cytokines, including IL-17, IL-23, and TNF-α. This immune activation contributes to systemic inflammation and may be shaped by microbial metabolites[15]

Gut Microbiota Alterations:

Studies have found differences in gut microbial diversity and composition between people with psoriasis and controls, including changes in Firmicutes and Bacteroidetes. Increased translocation of gut bacteria, such as *E. coli*, has also been associated with pro-inflammatory states[24].

Dietary Influences:

Anti-inflammatory eating patterns, such as Mediterranean diets rich in omega-3 fatty acids, antioxidants, and fiber, are associated with reduced disease severity and greater microbial diversity. In psoriatic models, high-fat Western diets worsen gut dysbiosis and IL-17-mediated inflammation[24].

5.3 Atopic Dermatitis (AD)

Pathophysiology:

AD is characterized by impaired epidermal barrier function and Th2-skewed immune responses, with cytokines and IgE among the key systemic factors involved[15].

Gut Microbiota Alterations:

People with AD often have less diverse gut microbiota, lower levels of beneficial bacteria such as *Bifidobacterium*, and fewer short-chain fatty acid (SCFA) producers. These changes are associated with disrupted immune regulation and greater inflammation[25].

Dietary Influences:

Fiber-rich, plant-based diets may support AD by promoting SCFA production and reducing gut inflammation, whereas Western dietary patterns may worsen dysbiosis[26].

5.4 Rosacea

Pathophysiology:

Rosacea is characterized by chronic facial flushing and inflammation involving neurovascular dysregulation and innate immune activation. Gut dysbiosis (e.g., small intestinal bacterial overgrowth) has been linked to rosacea symptomatology[27].

Gut Microbiota Alterations:

Although fewer gut microbiome studies exist for rosacea compared to other dermatoses, increased prevalence of GI dysbiosis and SIBO has been reported in patients with rosacea[27].

Dietary Influences:

Trigger foods (alcohol, spicy foods) can exacerbate symptoms. Anti-inflammatory diets and fermented foods may support microbial balance and reduce flares[27]'

5.5 Vitiligo

Pathophysiology:

Vitiligo is an autoimmune melanocyte-destructive condition involving CD8+ T cells and oxidative stress; systemic immune dysregulation and inflammatory mediators contribute to depigmentation[15].

Gut Microbiota Alterations:

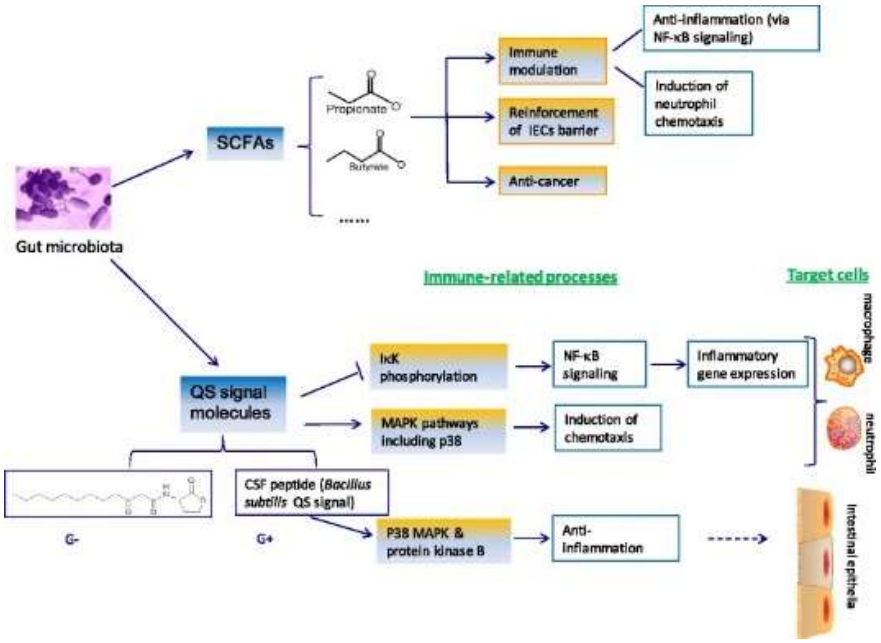
Preliminary evidence suggests altered gut microbial composition in vitiligo, potentially impacting systemic immune balance and oxidative status[15].

Dietary Influences:

Antioxidant-rich diets and polyphenol intake may ameliorate oxidative stress; gut-modulating diets could support immune regulation, but specific vitiligo dietary studies remain sparse[15].

6. Gut Microbial Metabolites and Their Relevance to Skin Health.

Fig 3. Role of intestinal microbiota and metabolites on gut homeostasis and human diseases (28)



Gut microbiota ferment dietary fibers to produce short-chain fatty acids (SCFAs) such as acetate, propionate, and butyrate. These SCFAs act as key signaling molecules by activating G-protein-coupled

receptors (GPCRs) on intestinal epithelial and immune cells. Through this signaling, SCFAs inhibit NF-κB and MAPK inflammatory pathways, leading to reduced expression of pro-inflammatory genes. They also promote regulatory immune responses, support controlled immune cell chemotaxis, and strengthen the intestinal epithelial barrier. Overall, SCFAs help maintain gut immune balance, reduce inflammation, and protect against gut-related diseases[28]

Mechanism of *Clostridioides difficile* Infection (CDI) in the Gut

1. Antibiotic-Induced Dysbiosis Enables Colonization

A healthy gut microbiota normally resists colonization by pathogens like *Clostridioides difficile* by competing for nutrients, producing inhibitory metabolites (e.g., secondary bile acids, SCFAs), and stimulating immune defenses. When broad-spectrum antibiotics disrupt this microbiota, colonization resistance is lost and *C. difficile* spores can germinate and proliferate[29]

2. Bile Acid Metabolism Drives Spore Germination

Gut bacteria normally convert primary bile acids (which promote *C. difficile* germination) into secondary bile acids (which inhibit germination and growth). Antibiotic-induced loss of bile-metabolizing bacteria increases primary bile acids like taurocholate, strongly promoting *C. difficile* spore germination in the intestine[30,31].

3. Toxins A and B Cause Epithelial Damage

Once vegetative *C. difficile* cells are established, they produce two main toxins—Toxin A (TcdA) and Toxin B (TcdB)—that enter host cells and disrupt cytoskeletal integrity by inactivating Rho GTPases. This leads to epithelial barrier breakdown, fluid secretion, and inflammation[32].

4. Inflammation and Clinical Disease

The epithelial damage and toxin effects trigger an immune response with neutrophil infiltration and cytokine release. The result is diarrhea, colitis, and, in severe cases, pseudomembranous colitis due to combined toxin action and local inflammation[33].

5. Persistence and Recurrence

Spores persist in the gut and environment; because antibiotics don't restore microbiota balance, recurrent CDI is common. Restoring gut microbiota (e.g., via fecal microbiota transplant) helps re-establish bile acid metabolism and inhibitory metabolites that suppress *C. difficile* growth[34].

7. Therapeutic Implications

- Consuming a healthy, plant-based diet can reduce skin inflammation and enhance overall skin health. Although the application of probiotics, synbiotics, and postbiotics has demonstrated promise in modulating inflammation, enhancing tissue regeneration, and inhibiting pathogenic colonization[26].
- Fecal microbiota transplantation represents a promising but still experimental strategy in psoriasis management by targeting gut dysbiosis and modulating systemic immune responses. While preliminary animal studies and limited human data suggest potential benefits in reducing inflammation and disease severity, robust clinical trials are required to establish its safety, efficacy, standardization, and long-term role in psoriasis care[35].
- Holistic approaches combine diet, lifestyle, and microbial modulation to support the gut–skin axis. These include:
  - Anti-inflammatory diets rich in fiber and phytonutrients
  - Fermented foods and prebiotic supplementation
  - Stress reduction and vitamin D optimization

Such integrative approaches aim to reduce systemic inflammation and strengthen both gut and skin barriers. Research reviews on microbial strategies discuss combining nutritional and microbial therapies to treat skin conditions effectively[26].

8. Current Evidence and Research Gaps

- **Limitations of existing studies:** Most research on the gut–skin axis in dermatologic diseases is limited by small sample sizes, cross-sectional designs, and heterogeneity in patient populations, which reduces the strength of causal inferences. Many studies focus on associations rather than mechanistic pathways.
- **Conflicting findings:** Variability in microbial alterations reported across studies (e.g., differences in Firmicutes/Bacteroidetes ratios, SCFA levels, and specific bacterial taxa) leads to inconsistent conclusions. Outcomes of dietary and probiotic interventions also show mixed efficacy, likely due to differences in study design, dosage, and patient characteristics.
- **Lack of standardized protocols:** There is no consensus on microbiome sampling, sequencing methods, analysis pipelines, or clinical endpoints. FMT procedures, probiotic formulations, and dietary interventions vary widely across studies, limiting reproducibility and comparability.
- **Need for longitudinal and interventional trials:** Few long-term or randomized controlled trials exist to clarify causality and therapeutic potential. Future studies should evaluate standardized dietary interventions, probiotics, and FMT in well-characterized cohorts, including assessment of biomarkers, clinical outcomes, and microbiome changes over time.
- **Integration into clinical practice:** Despite promising preliminary evidence, translating gut–skin axis research into standardized, personalized dermatologic therapies remains challenging. Defining predictive biomarkers and understanding inter-individual variability are critical research gaps that must be addressed.

9. Future Directions

- **Personalized nutrition:** Tailoring dietary interventions based on individual gut microbiome composition, metabolic profiles, and inflammatory status to optimize skin health and reduce disease severity.
- **Microbiome-based therapies:** Development of targeted probiotics, prebiotics, synbiotics, postbiotics, and standardized fecal microbiota transplantation protocols to restore microbial balance and modulate systemic and cutaneous inflammation.
- **Multi-omics approaches:** Integrating metagenomics, metabolomics, transcriptomics, and proteomics to better understand gut–skin interactions, identify biomarkers, and elucidate mechanisms linking microbiota, immune responses, and skin pathology.
- **Gut–brain–skin axis integration:** Investigating how neuroendocrine and psychoneuroimmune pathways interact with gut microbiota to influence skin inflammation, enabling combined interventions targeting diet, stress, and microbial modulation for holistic skin care.

10. Conclusion

- **Summary of key insights:** The gut–skin axis represents a complex, bidirectional network linking gut microbiota, immune regulation, neuroendocrine signaling, and skin health. Dysbiosis, barrier dysfunction, and altered microbial metabolites contribute to inflammatory and autoimmune skin

diseases such as psoriasis, atopic dermatitis, acne, rosacea, and vitiligo. Diet, probiotics, and microbiome-targeted interventions can modulate this axis to improve skin outcomes.

- **Clinical relevance:** Understanding the gut–skin axis provides opportunities for integrative dermatologic care, including dietary modification, microbial therapies, and lifestyle interventions aimed at reducing systemic inflammation and enhancing barrier function. Personalized strategies may improve disease management and patient quality of life.
- **Final perspective:** While evidence supporting gut–skin modulation is promising, further standardized, longitudinal, and mechanistic studies are needed. Integrating microbiome insights with diet, stress management, and immune-targeted therapies holds potential to transform dermatologic practice toward precision, holistic care.

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