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Research Article

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Epidermal Manifestations of Small Intestinal Bacterial Overgrowth and the Connection to Microbial Inflammation

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Abstract

Small intestinal bacterial overgrowth (SIBO), defined by the excessive proliferation of bacteria within the small intestine, has emerged as a significant contributor to systemic inflammation with downstream effects on cutaneous health. Evidence demonstrates a strong association between SIBO and dermatological conditions such as rosacea and atopic dermatitis, driven by shared pathophysiological mechanisms involving microbial dysbiosis, gut permeability, and systemic immune activation. Elevated gut-derived endotoxins, particularly lipopolysaccharides (LPS), trigger the release of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and interleukin-1β (IL-1β), exacerbating skin inflammation and disrupting epidermal homeostasis. Clinical data reveal that SIBO occurs in up to 38% of patients with rosacea and 24% of individuals with atopic dermatitis, which is significantly higher than in populations without SIBO. Therapeutic interventions targeting the gut microbiome have demonstrated significant efficacy; rifaximin therapy not only alleviates gastrointestinal symptoms but also reduces rosacea severity by up to 60%, likely through modulation of systemic inflammation. Probiotic supplementation has similarly shown promise by reducing the frequency and intensity of atopic dermatitis flares, potentially restoring gut microbial balance and improving skin barrier function. The gut-skin axis plays an integral role in the pathogenesis of inflammatory skin diseases, and microbiome-targeted therapies offer an opportunity to reimagine traditional approaches, creating a more holistic and effective framework for improving patient outcomes.

Introduction

Small intestinal bacterial overgrowth (SIBO) is defined by an excessive proliferation of bacteria in the small intestine, typically exceeding 10^5–10^6 organisms/mL [1]. The overgrowth of these organisms disrupts the normal balance of the gut microbiome, leading to intestinal symptoms like abdominal pain, bloating, and diarrhea and wide-ranging physiological consequences. SIBO affects up to 33.8% of patients with gastroenterological complaints, however, the actual number is likely higher due to asymptomatic cases and nonspecific symptoms [2]. SIBO often develops as a result of slowed orocecal transit time, which hinders the small intestine's ability to clear bacteria efficiently. This delay in transit can be caused by intestinal motility disorders, such as those seen in gastrointestinal conditions, diabetic autonomic neuropathy, and portal hypertension. Furthermore, a reduced stimulatory effect of thyroid hormones on intestinal motility may also contribute to bacterial overgrowth in the small intestine. Risk factors for SIBO include aging, the use of proton pump inhibitors, and the presence of comorbidities. Diseases associated with SIBO include irritable bowel syndrome, functional dyspepsia, chronic constipation, bloating, diarrhea, short bowel syndrome, lactase deficiency, diverticular disease, celiac disease, and ulcerative colitis, among many other GI disorders [2]. However, multiple sclerosis, autism, Parkinson's disease, systemic sclerosis, fibromyalgia, spondyloarthropathy, asthma, and heart failure have also been linked to SIBO. This highlights the broad spectrum of medical conditions that may contribute to or be caused by small intestinal bacterial overgrowth.

Small intestinal bacterial overgrowth (SIBO) has gained attention as a key player in systemic inflammation and its broader effects on health, particularly cutaneous health. The link between gut health and skin health is well-recognized, with many GI disorders presenting with skin-related symptoms [3]. One of the most compelling areas of research highlights its impact on skin conditions such as rosacea and atopic dermatitis. Pertaining to skin microbiota, rosacea is often associated with *demodex folliculorum* mites, whereas atopic dermatitis is associated with decreased bacterial diversity and the increased abundance of *Staphylococcus aureus* [4,5]. This paper explores the multifaceted connections between small intestinal bacterial overgrowth and cutaneous inflammation, underscoring shared mechanisms, clinical associations, and therapeutic strategies.

Review

Pathophysiology of Small Intestinal Bacterial Overgrowth

At the heart of small intestinal bacterial overgrowth's (SIBO) pathology lies microbial dysbiosis, where the overgrowth of certain bacterial species in the small intestine disrupts normal gut function [6]. Often, this dysbiosis represents an increase in

gram-negative bacteria, leading to the overproduction of endotoxins, particularly lipopolysaccharides (LPS), that compromise intestinal barrier integrity [7]. This occurs through LPS binding toll-like receptor 4 (TLR4) on intestinal epithelial cells, causing the disruption of tight junctions by contractions of the perijunctional actomyosin ring. Similarly, another mechanism in which dysbiosis leads to disruption of tight junctions involves reduced production of short-chain fatty acids (SCFAs), which causes decreased expression of tight junction proteins [8]. Tight junctions are responsible for creating a seal between intestinal cells, and disrupting them causes increased gut permeability, often referred to as "leaky gut." This allows bacterial components and toxins to enter the bloodstream, triggering systemic immune responses. The cascade of inflammation that follows is believed to play a significant role in the exacerbation of skin diseases.

The gut-skin axis represents a bidirectional communication network linking gastrointestinal health to skin homeostasis [9,10]. In SIBO, the breach in gut barrier integrity allows pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and interleukin-1β (IL-1β) to circulate systemically. These inflammatory mediators disrupt skin homeostasis by altering keratinocyte function, reducing barrier integrity, and increasing skin sensitivity to environmental triggers. In addition, the chronic inflammatory state introduced by these skin conditions worsens dysbiosis, which leads to further destruction of the intestinal barrier, demonstrating a vicious cycle. This interplay between gut-derived inflammation and epidermal dysfunction positions small intestinal bacterial overgrowth as a key contributor to skin diseases such as rosacea and atopic dermatitis.

SIBO and Rosacea: Clinical and Pathogenic Association

Rosacea is one of the most well-studied dermatological conditions associated with small intestinal bacterial overgrowth. Studies have reported that up to 46% of patients with rosacea also have underlying SIBO, significantly higher than in control populations [11]. The chronic inflammation characteristic of rosacea mirrors the systemic immune activation seen in SIBO, suggesting a shared pathogenic pathway. Bacteria most commonly linked to rosacea include *Helicobacter pylori*, *Staphylococcus epidermidis*, *Chlamydia pneumoniae*, and *Bacillus oleronius* [12]. The mechanism in which these inflammatory markers cause rosacea is not fully understood, but one proposition links *Bacillus oleronius* to rosacea via heightened angiogenesis driven by increased production of cathelicidin and kallikrein 5 peptides, which promotes type 1 IFNs [13]. Moreover, the reduction in rosacea severity following rifaximin therapy, a treatment for small intestinal bacterial overgrowth, supports the gut-skin axis hypothesis, highlighting the microbiome's role in skin inflammation [11].

Atopic Dermatitis and Gut Dysbiosis

Atopic dermatitis (AD), a chronic inflammatory skin condition, is another disease linked to gut microbial changes [14]. The connection is underpinned by gut permeability and systemic inflammation, both of which exacerbate the cytokine dysregulation seen in AD. The "microflora hypothesis of allergic disease" states that infants who are exposed to gut microbes are more likely to shift away from T-helper 2 cells (Th2 cells). Th2 cells release cytokines which lead to immunoglobulin E (IgE) production and atopic conditions such as AD, allergies and asthma. Reddel et al. found that in children with atopic dermatitis, there was a reduction of SCFA bacteria in the gut microbiota and increase in *Faecalibacterium*, *Oscillospira*, *Bacteroides*, *Parabacteroides* and *Sutterella* species [14]. Probiotic therapies have shown potential in AD management by restoring gut microbial balance and reducing inflammation, further emphasizing the gut-skin axis's role [15]. The probiotics shown to have the best effect in reducing skin lesions include formulas with *Bifidobacterium* and *Lactobacillus* species. The main way in which these probiotics help improve eczema is by way of improving the intestinal barrier.

Disruption of Epidermal Homeostasis: Impact of SIBO-Induced Systemic Inflammation

The endotoxins produced in small intestinal bacterial overgrowth, particularly lipopolysaccharides, play a pivotal role in driving systemic inflammation [7]. LPS binds to toll-like receptor 4 (TLR4) on immune cells, triggering the release of pro-inflammatory cytokines including interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and interleukin-1β (IL-1β). This cascade not only worsens systemic inflammation but also directly impacts skin health [10,16]. Increased cytokine levels disrupt the skin barrier through disrupting tight junctions and impairs epidermal repair mechanisms. This causes the skin to be more sensitive to allergens and other pathogens, leading to intensified inflammatory responses in conditions such as rosacea and atopic dermatitis.

Systemic immune activation in SIBO creates a pro-inflammatory milieu resulting in increased intestinal permeability. Intestinal bacteria and cytokines undergo systemic distribution traveling to distant organs, including the skin [17]. Elevated levels of TLR-4, IL-6, TNF-α and IL-1β disrupt epidermal homeostasis by impairing keratinocyte differentiation and reducing lipid production [18,19]. The compromised barrier function increases transepidermal water loss, heightening the skin's susceptiblity to environmental irritants, allergens, and microbial invasion. Chronic inflammation associated with SIBO may also exacerbate pre-existing skin conditions, such as psoriasis and atopic dermatitis, by amplifying immune dysregulation [20-22]. Additionally, systemic inflammation may influence the skin microbiome, altering its composition and potentially promoting dysbiosis. Understanding this gut-skin axis highlights the importance of addressing SIBO as a potential therapeutic target for managing inflammatory skin diseases, emphasizing the interplay between gastrointestinal health and dermatologic outcomes.

Integrative Treatment Strategies

Small intestinal bacterial overgrowth requires a multifaceted treatment approach, incorporating antibiotics, supplements, and dietary changes. Rifaximin, a non-absorbable antibiotic, has shown particular effectiveness in treating SIBO by significantly reducing systemic inflammation and improving certain skin conditions, such as rosacea [17]. One study found that eradicating SIBO with rifaximin significantly improved cutaneous lesions among rosacea patients, with 20 out of 28 experiencing complete clearance of their skin lesions [11]. This notable improvement underscores the connection between decreased gut inflammation and reduced systemic inflammation, highlighting the potential of SIBO-targeted treatments as valuable adjuncts in dermatological care. In contrast, other antibiotics like metronidazole, ciprofloxacin, and amoxicillin-clavulanate are used to treat SIBO based on clinical

experience rather than robust evidence [23]. These alternatives have proven less effective than rifaximin for SIBO treatment and lack validation for improving skin conditions. This knowledge gap suggests a promising avenue for future research to explore the gut-skin axis further.

In addition to antibiotics, less aggressive options are being explored to mitigate gut bacterial overgrowth and enhance dermatological outcomes. Advances in microbiome research have identified specific bacterial species associated with SIBO, paving the way for personalized therapeutic strategies. Prebiotics, probiotics, postbiotics, and synbiotics are emerging as practical tools for modulating the gut microbiome and potentially improving gastrointestinal and skin health. For example, prebiotics like fructooligosaccharides (FOS) can promote beneficial gut bacteria such as *Staphylococcus epidermidis* while inhibiting pathogenic species like *Staphylococcus aureus*. This process involving FOS produces short-chain fatty acids (SCFAs), which enhance gut health and reduce systemic inflammation [24]. Probiotic supplementation with strains like *Lactobacillus plantarum* and *Lactobacillus rhamnosus* has also shown promise for managing SIBO-related skin issues [25]. Clinical trials indicate that probiotics can reduce the frequency of atopic dermatitis flares by modulating immune responses and supporting skin barrier function. Moreover, postbiotics like butyrate have demonstrated the potential to maintain skin health through inflammatory modulation [26]. This integrated approach aims to restore microbial balance and reduce systemic inflammation by combining antibiotic treatment with prebiotics, probiotics, and postbiotics.

Dietary modifications also play a critical role in managing small intestinal bacterial overgrowth over the long term. Low-Fermentable Oligo-, Di-, Mono-saccharides, And Polyols (FODMAP) diets have effectively reduced intestinal pathogenic bacterial overgrowth [27]. A low-FODMAP diet minimizes fermentable carbohydrates in foods such as garlic, onions, sugar-free candy, breakfast cereals, cow's milk, and grapes [28]. By reducing pathogenic fermentation in the gut, these diets lower hydrogen production and endotoxin levels, decreasing local and systemic inflammation and SIBO symptoms. However, the restrictive nature of low-FODMAP diets can lead to deficiencies in essential nutrients like fiber, calcium, iron, zinc, folate, and vitamins B and D if not carefully managed [29]. To mitigate these risks, collaboration with a dietician is beneficial. Implementing a phased approach—consisting of elimination, reintroduction, and personalization—can also help individuals identify specific triggers while maintaining a nutrient-rich diet [30]. Overall, a well-managed low-Fermentable Oligo-, Di-, Mono-saccharides, And Polyols diet, guided by a dietitian, effectively manages small intestinal bacterial overgrowth while also improving skin conditions by reducing inflammation and promoting gut health.

Finally, alternative therapies are being investigated for their potential to enhance gastrointestinal motility and improve both small intestinal bacterial overgrowth symptoms and skin health. Prokinetic agents like metoclopramide and erythromycin show promise in preventing bacterial overgrowth by enhancing gut motility [31]. Notably, a regimen including Biocidin liquid tincture and GI Detox+ demonstrated an improvement in SIBO symptoms and a 20% reduction in facial erythema after ten weeks by targeting hydrogen sulfide-dominant gut bacteria [32]. Additionally, herbal remedies such as berberine and oregano oil possess antimicrobial properties that may alleviate SIBO symptoms [33]. While these alternative therapies present exciting possibilities for treatment enhancement, caution is warranted. Recent systematic reviews highlight the need for more extensive randomized controlled trials to fully validate these alternative therapies' efficacy [27,33]. The advancements in small intestinal bacterial overgrowth treatment options present a hopeful outlook for future research focused on developing personalized therapeutic interventions that can enhance patient outcomes internally and externally.

Table 1: Therapeutic Strategies Targeting SIBO and Their Impact on Cutaneous Health.

Intervention	Mechanism of Action	Impact on Skin Health	Clinical Evidence
Rifaximin	Non-absorbable antibiotic; reduces small bowel bacterial load	Decreases systemic inflammation; improves rosacea severity	20 of 28 rosacea patients had complete skin lesion clearance after treatment [11]
Probiotics (e.g., *Lactobacillus*, *Bifidobacterium*)	Restore microbial balance; promote intestinal barrier repair	Reduce flares in atopic dermatitis; enhance skin barrier function	Modulate immune responses; supported in pediatric AD trials [14,15]
Prebiotics (e.g., FOS)	Stimulate growth of beneficial bacteria; increase SCFA production	Indirectly reduce systemic inflammation and enhance skin resilience	FOS suppresses *S. aureus*, promotes *S. epidermidis* [24]
Postbiotics (e.g., Butyrate)	Direct metabolic products of beneficial microbes; anti-inflammatory	Enhance epithelial repair and reduce immune activation in skin	Experimental models show improved epidermal barrier function [26]
Low-FODMAP Diet	Reduces fermentable substrates for bacteria, lowering gas and endotoxin production	Decreases hydrogen production and skin flare severity in SIBO patients	Requires dietitian guidance to avoid nutrient deficiency [27–30]
Prokinetic Agents (e.g., Metoclopramide)	Enhance GI motility to prevent bacterial stasis	May reduce systemic inflammatory load affecting skin	Supportive data in SIBO prevention but limited on cutaneous effects
Herbal/Nutraceutical Agents (e.g., Berberine, Oregano Oil)	Antimicrobial and anti-inflammatory properties	Potential skin benefit through SIBO symptom relief	Preliminary findings; more clinical trials needed [33]

Challenges and Future Directions

Accurately diagnosing small intestinal bacterial overgrowth remains a challenge due to the limitations of current testing methods, such as breath tests and jejunal aspirates. These methods often lack sensitivity and specificity leading to misdiagnosis. This can lead to inappropriate treatments, such as unnecessary antibiotics or dietary restrictions, which may exacerbate symptoms. Furthermore, prolonged systemic inflammation associated with undiagnosed or poorly managed SIBO may contribute to flares of conditions like rosacea, atopic dermatitis and psoriasis through the gut skin axis. Advances in diagnostic tools like molecular and microbiome analyses offer the potential for more precise identifications of SIBO and its subtypes [34]. These emerging tools are crucial for enabling more targeted interventions thus improving patient outcomes.

Emerging therapies, including microbiome transplantation and phage therapy, offer exciting possibilities for treating SIBO and its associated conditions. Fecal microbiota transplantation (FMT) involves the transplant of stool from a healthy donor into the gastrointestinal tract of the patient with SIBO. FMT has been demonstrated to restore the normal gut microbiota in individuals with SIBO. However, the routine use of FMTs for SIBO has not been tested due to feasibility issues [35]. Bacteriophage therapy utilizes bacteriophages, viruses that infect bacteria, to target certain bacterial species to restore the microbial gut equilibrium. While a promising therapeutic, bacteriophage therapy is still a novel technique and still requires Phase II trials in mammalian hosts as well as phage and bacterial dynamics studies [36]. These innovative approaches aim to reestablish microbial equilibrium in the gut, potentially reducing systemic inflammation and improving skin health. Continued research into these therapies could revolutionize the management of SIBO-related diseases.

The interplay between SIBO and skin health underscores the importance of the gut-skin axis in understanding and managing inflammatory skin conditions. By addressing the microbial and inflammatory drivers of diseases such as rosacea and atopic dermatitis, SIBO-targeted therapies offer a transformative approach to improving patient outcomes. The growing body of evidence linking gut and skin health highlights the need for a holistic perspective in treating chronic inflammatory diseases, paving the way for innovative, microbiome-focused interventions.

Conclusion

The connection between small intestinal bacterial overgrowth (SIBO) and cutaneous health highlights the critical importance of the gut-skin axis in understanding and managing inflammatory skin conditions. SIBO disrupts microbial balance and increases gut permeability, initiating systemic immune activation characterized by elevated lipopolysaccharides (LPS) and pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and interleukin-1β (IL-1β). The immune cascade not only amplifies gastrointestinal symptoms but also impairs epidermal homeostasis, contributing to skin barrier dysfunction and chronic inflammation seen in conditions like rosacea and atopic dermatitis. Clinical studies demonstrate that therapies targeting the gut microbiome, such as rifaximin, significantly alleviate gastrointestinal symptoms and reduce the severity of dermatological manifestations, with improvements of up to 60% reported in rosacea patients. Probiotic supplementation further supports skin health by restoring microbial balance, reducing inflammation, and enhancing skin barrier repair mechanisms. The growing focus on personalized interventions, including tailored dietary strategies and advanced microbiome modulation techniques, offers a promising path for mitigating the microbial and inflammatory drivers of SIBO-related skin diseases. A comprehensive approach addressing the systemic origins of these conditions provides an opportunity for long-term improvements in both gastrointestinal and dermatological outcomes. By integrating gut-focused therapies into dermatological care, clinicians can address the underlying causes of inflammation and microbiome disruption, ultimately advancing the standard of care for patients with chronic inflammatory skin disorders.

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