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The Gut-Skin Axis in Rosacea: Beyond Facial Flushing - A Clinical Review of SIBO, Microbiome Dysbiosis, and Systemic Management

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Abstract

Background: Rosacea has traditionally been managed as an isolated chronic dermatological disorder. However, emerging clinical evidence over the last decade highlights a profound systemic link between intestinal dysbiosis and cutaneous neurovascular inflammation, mediated by the gut-skin axis. Objective: This review synthesizes recent clinical data regarding the pathophysiological mechanisms, epidemiological associations, and management strategies linking small intestinal bacterial overgrowth (SIBO), gastrointestinal (GI) comorbidities, and rosacea.

Methods: A review of literature from baseline clinical trials through recent data was conducted, evaluating the molecular bridges, colonic dysbiosis, and systemic therapeutic interventions in refractory rosacea.

Results: Epidemiological data demonstrate a stark contrast in SIBO prevalence, affecting 46% to 52% of rosacea patients compared to a much lower baseline in the general population. The overgrowth of colonic-type bacteria yields hydrogen (H2) and methane (CH4) gases, causing mucosal barrier breakdown ("leaky gut"). This drives the systemic translocation of microbial antigens, generating reactive oxygen species (ROS) and pro-inflammatory cytokines (TNF-α, IL-1 beta, IL-6) that trigger cathelicidin activation and facial neurovascular hyperreactivity. Targeted SIBO eradication with the gut-selective antibiotic rifaximin yields complete cutaneous lesion clearance in up to 78% of patients, with long-term remission spanning up to three years.

Conclusion: Refractory rosacea frequently serves as a cutaneous manifestation of underlying GI pathology. Integrating dermatological interventions with a "gut-first" approach incorporating rifaximin, multi-strain probiotics, and low-FODMAP dietary modifications establishes a comprehensive multidisciplinary care model capable of achieving true, systemic disease resolution.

Keywords: Gut-skin axis; Small intestinal bacterial overgrowth (SIBO); Rifaximin; Gastrointestinal; Inflammatory

Abbreviations: GI: Gastrointestinal; IBD: Inflammatory Bowel Disease; IBS: Irritable Bowel syndrome; SIBO: Small Intestinal Bacterial Overgrowth; SCFAs: Short-Chain Fatty Acids; TRP: Transient Receptor Potential; H2: Hydrogen Gas; CH4: Methane Gas; LPS: Lipopolysaccharides; TLR4: Toll-Like Receptor 4; ROS: Reactive Oxygen Species; TNF-alpha: Tumor Necrosis Factor-Alpha; IL-1beta: Interleukin-1 Beta; IL-6: Interleukin-6; GWAS: Genome-Wide Association Study; HLA: Human Leukocyte Antigen; HPA: Hypothalamic-Pituitary-Adrenal; BDNF: Brain-Derived Neurotrophic Factor; PDL: Pulsed Dye Laser; KTP: Potassium-Titanyl-Phosphate; FODMAP: Fermentable Oligosaccharides, Disaccharides, Monosaccharides, And Polyols; TLR2: Toll-Like Receptor 2; MyD88: Myeloid Differentiation Primary Response 88; NF-kappaB: Nuclear Factor Kappa-Light-Chain-Enhancer of Activated B Cells

Introduction

Rosacea has traditionally been considered a chronic inflammatory disorder characterized mainly by facial erythema, flushing, papules, pustules, and telangiectasia. However, growing evidence over the last decade has expanded the understanding of rosacea beyond an isolated dermatologic condition, recognizing it as a complex neurovascular and inflammatory disorder involving dysregulation of innate immunity, vascular hyperreactivity, and chronic inflammation [1,2]. Current evidence suggests that interactions between the immune system, cutaneous vasculature, nervous system, and intestinal microbiome contribute significantly to disease development and progression [2,3]. This evolving concept has led to increasing interest in the gut–skin axis as a potential mechanism linking gastrointestinal (GI) dysfunction with cutaneous inflammation in rosacea patients [3].

Historically, GI symptoms in patients with rosacea were often regarded as coincidental findings. Nevertheless, recent epidemiological studies have demonstrated a significantly higher prevalence of GI comorbidities among rosacea patients compared with the general population, including inflammatory bowel disease (IBD), irritable bowel syndrome (IBS), celiac disease, Helicobacter pylori infection, and small intestinal bacterial overgrowth (SIBO) [3,4]. These findings have further supported the hypothesis that chronic intestinal inflammation and alterations in the gut microbiome may contribute to immune activation and disease exacerbation. Increased intestinal permeability, microbial metabolite translocation, and cytokine release from the gastrointestinal tract have been proposed as possible mechanisms promoting neurovascular dysregulation and persistent cutaneous inflammation in rosacea [5].

More recent evidence published between 2024 and 2026 has further reinforced the association between rosacea, intestinal dysbiosis, and SIBO [6,7]. Microbiome and metagenomic sequencing studies have identified reduced microbial diversity and alterations in bacterial composition in rosacea patients, including decreased production of anti-inflammatory metabolites such as short-chain fatty acids (SCFAs) [6]. In addition, several studies have reported a higher prevalence of SIBO in individuals with rosacea, with clinical improvement observed after eradication therapy using rifaximin, supporting a possible pathogenic role of bacterial overgrowth in the development and persistence of cutaneous inflammation [7,8]. Although the precise causal relationship between dysbiosis and rosacea remains incompletely understood, current evidence increasingly supports the concept of rosacea as a multifactorial disorder influenced by complex interactions between the gastrointestinal tract, immune system, nervous system, and skin.

Pathophysiology: The Molecular Bridge

The pathophysiology of rosacea may be understood as a molecular connection between intestinal inflammation, innate immune activation, and facial neurovascular reactivity. In patients with rosacea, dysregulation of innate immunity is closely linked to abnormal activation of the kallikrein-5 and cathelicidin pathway. Increased kallikrein-5 activity promotes the processing of cathelicidin into its active peptide form, LL-37, which exerts pro-inflammatory and vasoactive effects in facial skin [9,10]. Within the context of the gut–skin axis, intestinal inflammation and microbial imbalance may amplify systemic immune signaling, potentially contributing to this cutaneous inflammatory cascade and worsening facial erythema, papules, and persistent inflammation [11,12].

Neurovascular dysregulation also plays an important role in rosacea. Transient receptor potential channels, particularly TRPV1 and TRPA1, function as sensory receptors involved in heat perception, pain, flushing, and neurogenic inflammation [13]. These channels may be activated or sensitized by common rosacea triggers such as heat, spicy foods, alcohol, and inflammatory mediators, leading to the release of neuropeptides that promote vasodilation and facial flushing [13,14]. More recent gut–skin axis models suggest that bacterial metabolites and intestinal dysbiosis may further influence neuroimmune signaling pathways, although the direct relationship between gut-derived metabolites and TRP channel activation in rosacea remains under investigation [11,12].

Increased intestinal permeability, commonly referred to as "leaky gut," may provide another mechanism linking gastrointestinal inflammation with facial disease activity. Disruption of the intestinal barrier may allow microbial products, metabolites, and inflammatory cytokines to enter systemic circulation and contribute to chronic low-grade immune activation [11,15]. This cytokine spillover may subsequently affect the facial microvasculature, promoting endothelial activation, neurovascular sensitivity, and persistent cutaneous inflammation. Although these mechanisms are biologically plausible and increasingly supported by gut–skin axis research, additional longitudinal and mechanistic studies are still needed to clarify their precise role in rosacea pathogenesis [11,12,15].

The Role of SIBO (Small Intestinal Bacterial Overgrowth)

Epidemiological Links

The pathophysiological framework of the gut-skin axis suggests that localized intestinal disruptions can manifest as systemic, cutaneous inflammatory pathologies [16]. Among these disruptions, SIBO has emerged as a significant disease-modifying factor rather than a mere coincidental comorbidity [16]. Epidemiological data demonstrate a stark contrast in SIBO prevalence between patients with rosacea and the general population [17]. In the general population, the baseline prevalence of SIBO is relatively low, typically estimated to range between 2.5% and 22% depending on the diagnostic modalities utilized [17]. Conversely, patients diagnosed with rosacea display significantly higher rates of bacterial overgrowth, with landmark case-control data indicating a SIBO prevalence of approximately 46% to 52% within the rosacea cohort [17,18]. This disparity

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underscores a robust epidemiological association, suggesting that a compromised or permissive small intestinal environment is disproportionately prevalent in individuals with the neurovascular and inflammatory phenotypes of rosacea [16,18].

Metabolic Byproducts

The migration and excessive proliferation of colonic-type bacteria into the upper gastrointestinal tract generate significant quantities of metabolic byproducts, primarily hydrogen (H2) and methane (CH4) gas [19]. While these gases cause direct mucosal injury, local distension, and altered gastrointestinal motility, their systemic ramifications drive the cutaneous pathology of rosacea [19,20]. The persistent metabolic activity of an overgrown microbiome triggers an up-regulation of localized and systemic immune responses [20]. This intestinal dysbiosis promotes increased intestinal permeability (leaky gut syndrome), which facilitates the translocation of bacterial products like lipopolysaccharides (LPS) into the bloodstream [16].

The systemic circulation of these microbial antigens activates toll-like receptors (such as TLR4) on circulating macrophages and endothelial cells, initiating a cascade that generates excessive reactive oxygen species (ROS) and releases pro-inflammatory cytokines like tumor necrosis factor-alpha (TNF-alpha), interleukin-1 beta (IL-1beta), and interleukin-6 (IL-6) [16]. The resulting systemic oxidative stress impairs neurovascular regulation and amplifies aberrant innate immune processing-such as cathelicidin activation—within the skin, ultimately translating abdominal dysbiosis into persistent facial erythema, papules, and pustules [16].

Clinical Evidence

The most compelling proof-of-concept validating the clinical relevance of the gut-skin axis in rosacea is the dramatic clearance of dermatological symptoms following the targeted eradication of SIBO [16,18]. In randomized clinical trials, rosacea patients who tested positive for SIBO via hydrogen and methane breath tests were treated with Rifaximin-a non-absorbed, gut-directed, oral antibiotic [18,20]. Rifaximin is uniquely suited for this therapeutic niche because its minimal systemic absorption concentrates its antimicrobial and localized anti-inflammatory effects entirely within the gastrointestinal lumen [20]. Following a standard therapeutic regimen of Rifaximin (e.g., 1200 mg/day for 10 days), clinical trials reported a SIBO eradication rate that directly correlated with objective cutaneous improvement [18].

Specifically, approximately 71% to 78% of SIBO-eradicated patients achieved complete clearance of their active facial rosacea lesions, with an additional 17% demonstrating a marked reduction in inflammatory severity [18,20]. In stark contrast, SIBO-positive patients randomized to receive a placebo experienced no clinical change or worsening of their skin condition [18]. Furthermore, long-term follow-up studies confirm that this response is durable, with up to 96% of successful responders remaining entirely symptom-free at 9 months, and 65% maintaining clinical remission at 3 years [20]. This high rate of therapeutic success establishes SIBO eradication as a powerful systemic management strategy for refractory rosacea [18,20].

Microbiome Dysbiosis and Gut Health

Helicobacter pylori Involvement

Helicobacter pylori has been increasingly recognized as a possible factor involved in the development and exacerbation of rosacea due to the close relationship between gastrointestinal health and inflammatory skin diseases. Several studies have reported a higher frequency of H. pylori infection in rosacea patients compared to healthy individuals, although the association remains controversial and requires further investigation [21,22]. Different mechanisms have been proposed to explain this relationship, including systemic inflammation, increased oxidative stress, and the release of pro-inflammatory cytokines and vasoactive mediators that may contribute to facial erythema and flushing [21]. In addition, H. pylori infection may alter gut microbiota balance and immune regulation, supporting the concept of the gut–skin axis in rosacea pathophysiology [23]. Interestingly, some studies have also shown clinical improvement of rosacea symptoms after eradication therapy, suggesting that gastrointestinal dysbiosis may play a role in disease progression [21].

Colonic Dysbiosis and Short-Chain Fatty Acids

Recent evidence has highlighted the important role of colonic microbiome alterations in the pathogenesis of rosacea. Several studies have demonstrated that patients with rosacea exhibit reduced gut microbial diversity compared to healthy individuals, suggesting the presence of intestinal dysbiosis and disruption of normal microbial homeostasis [24]. This loss of microbial diversity may impair the balance between beneficial and pro-inflammatory bacterial species, contributing to chronic low-grade systemic inflammation and abnormal immune activation, both of which are believed to play a significant role in inflammatory skin diseases. In addition, alterations in the colonic microbiota may affect intestinal barrier integrity and promote increased intestinal permeability, allowing inflammatory mediators and bacterial metabolites to enter systemic circulation and potentially influence cutaneous inflammation through the gut–skin axis [24,25].

Another important finding in rosacea patients is the reduction of short-chain fatty acids (SCFAs), particularly butyrate, which is produced by beneficial gut bacteria during the fermentation of dietary fiber [25]. Butyrate is known to exert multiple protective effects within the gastrointestinal tract, including maintenance of epithelial barrier function, modulation of immune responses, regulation of T-cell activity, and suppression of pro-inflammatory cytokine production [25]. Therefore, decreased levels of butyrate and reduced abundance of butyrate-producing bacteria may contribute to immune dysregulation and persistent systemic inflammation observed in rosacea patients. Furthermore, diminished SCFA production may weaken intestinal homeostasis

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and further exacerbate the inflammatory pathways involved in rosacea pathophysiology [24,25].

Metagenomic Insights

Advances in metagenomic sequencing have provided deeper insight into the composition of fecal microbiota in patients with rosacea, further supporting the role of the gut–skin axis in disease pathogenesis. Metagenomic analysis allows comprehensive identification of microbial populations and functional alterations within the intestinal microbiome, including bacterial species that cannot be detected through conventional culture methods. Several studies have demonstrated significant differences in the fecal microbiota of rosacea patients compared to healthy controls, including alterations in bacterial abundance and diversity associated with inflammatory and metabolic pathways. These microbial changes may contribute to immune dysregulation, increased intestinal permeability, and chronic systemic inflammation, which are considered important mechanisms involved in rosacea development. In addition, metagenomic sequencing studies have suggested that alterations in microbial metabolites and bacterial functional activity may influence host immune responses and promote cutaneous inflammation through complex interactions between the gastrointestinal tract and the skin [26].

Internal Medicine Implications: Systemic Comorbidities

Apart from its cutaneous manifestations, rosacea is associated with significant gastrointestinal and neuropsychiatric comorbidities, highlighting its systemic involvement. Numerous meta-analyses have confirmed a bidirectional relationship with inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis. The metabolic and inflammatory connections between rosacea and gut pathology are well documented [27,28]. Patients with rosacea are far more likely to acquire IBD, with risk ratios ranging from 1.18 to 1.58, while those with preexisting IBD have an elevated prevalence of rosacea compared to control populations [27,28]. This bidirectional relationship likely indicates shared pathophysiological mechanisms, such as the dysregulation of innate and adaptive immunity via Toll-like receptor 2 (TLR2) activation, dysregulation of mast cell functions, and the elevated production of pro-inflammatory cytokines like tumor necrosis factor (TNF) and IL-17 [29].

Another important gastrointestinal association is celiac disease; there is a twofold increased odds of celiac disease in rosacea patients, as demonstrated in a large population-based study [30]. Genome-wide association studies (GWAS) have identified shared genetic risk loci, including HLA-DQB1*02:01 and HLA-DQA1*05:01, pointing to a common immunogenetic predisposition [29,30]. Furthermore, according to recent cohort data, there is a 46% higher chance of developing irritable bowel syndrome (IBS) in rosacea patients, which may be mediated by systemic inflammatory pathways involving TNF and the gut-skin axis [31,32]. The neurologic connection between rosacea and psychiatric diseases, especially depression, has emerged as a major clinical concern. Meta-analyses show that patients with rosacea have a markedly increased risk of anxiety and depression, and Mendelian randomization studies have further supported this causal association [33,34].

Elevated pro-inflammatory cytokines and neuropeptides in rosacea patients disrupt neuroendocrine-immune interactions and promote neuroinflammation, suggesting a clear physiological link between neuroimmune dysregulation and chronic inflammation [33,35]. In addition, stress-induced dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis could worsen the inflammatory process through cortisol-mediated pathways, aggravating cutaneous flushing and depressive episodes in a bidirectional loop [33,35]. This neurobiological link between chronic facial flushing and mood disorders is further supported by lower levels of serum brain-derived neurotrophic factor (BDNF), especially in rosacea patients with comorbid depression [35]. These findings highlight the critical need for screening rosacea patients for GI and psychiatric symptoms, establishing the gut-brain-skin axis as a vital framework to develop new therapies targeting systemic inflammation rather than isolated cutaneous manifestations [29,35,36].

Integrative Management Strategies

The management of rosacea increasingly focuses on an integrative approach that pairs conventional dermatologic interventions with gut-targeted treatments and dietary modifications, reflecting the role of the gut-skin axis. Standard dermatological care remains fundamental; topical brimonidine tartrate 0.33% gel provides rapid vasoconstriction via selective alpha2-adrenergic receptor agonism to treat persistent erythema and flushing, while topical ivermectin 1% cream targets both Demodex mite burden and inflammatory pathways in papulopustular rosacea [37,38]. Vascular lasers are an indispensable tool for treating refractory telangiectasias and persistent erythema; pulsed dye lasers (PDL) operating at 585–595 nm wavelengths and potassium-titanyl-phosphate (KTP) lasers at 532 nm achieve selective photothermolysis of dilated facial vessels and are frequently paired with brimonidine for increased efficacy [39,40].

Since gut dysbiosis has been identified as a contributing factor, systemic adjuvants that target the intestinal microbiota have been introduced into treatment algorithms. One such adjuvant is rifaximin, a gut-selective, minimally absorbed antibiotic that has shown remarkable efficacy in rosacea patients with concurrent SIBO [41,42]. A standard course of rifaximin therapy (1200 mg daily for 10 days) eradicated SIBO in 87.5% of treated patients, yielding complete resolution of cutaneous lesions in 78% and sustained remission for at least nine months, according to landmark studies by Parodi and colleagues [41,42]. This clinical success suggests that intestinal dysbiosis-driven systemic inflammation can amplify facial inflammatory cascades. Rifaximin, with its targeted

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intestinal activity and negligible systemic absorption, treats the gut microbiota rather than the cutaneous flora as its therapeutic target, reinforcing the validity of the gut-skin axis [41].

Because certain strains like Lactobacillus rhamnosus have demonstrated an ability to enhance skin barrier functions, modify systemic inflammation, and influence neuroimmune pathways, probiotics and prebiotics have become compelling adjunctive therapies. Consequently, they have been termed "psychodermabiotics"-agents capable of addressing both the cutaneous and neuropsychiatric manifestations of rosacea [43-46]. When combined with conventional antibiotic therapy, recent clinical trials using multi-strain probiotic formulations containing Lacticaseibacillus rhamnosus, Lactiplantibacillus plantarum, and Bifidobacterium species have shown significant improvements in both gut microbiota composition and rosacea severity [47].

Dietary interventions further complement these microbial strategies. Low-FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols) diets may lower the metabolic substrate burden of SIBO and reduce related systemic inflammation, thereby decreasing the frequency of cutaneous flare-ups [48,49].

Conversely, diets high in fiber and rich in omega-3 polyunsaturated fatty acids (derived from sources like flaxseeds and fatty fish) exert distinct anti-inflammatory effects by inhibiting pro-inflammatory cascades, specifically the TLR2/MyD88/NF-kappaB pathway [51]. Randomized controlled trials have documented measurable improvements in the symptoms of ocular rosacea following omega-3 supplementation [50-52]. By combining topical vasoconstrictors and antimicrobials, laser-based vascular ablation, gut-directed antibiotics, probiotic supplementation, and anti-inflammatory nutritional modifications, these multimodal approaches represent a fundamental shift away from treating rosacea as an isolated dermatologic condition and towards dealing with it as a systemic inflammatory condition mediated by gut-brain-skin axis dysregulation [53].

Future Directions and Conclusion

The Mandate for Multidisciplinary Care

The evolving paradigm of the gut-skin axis necessitates a shift from isolated dermatological treatments to a comprehensive, multidisciplinary care model [1,3]. Historically, the management of rosacea has focused primarily on topical interventions, vascular lasers, and systemic anti-inflammatory agents targeted exclusively at cutaneous symptoms [2,39]. However, given the significant epidemiological overlap between rosacea and underlying gastrointestinal pathologies like SIBO, inflammatory bowel disease, and Helicobacter pylori infections, dermatologists can no longer view the skin in isolation [4,22,28].

Clinicians must actively screen patients with rosacea for comorbid gastrointestinal symptoms, such as chronic bloating, altered bowel habits, abdominal pain, and dyspepsia, during initial evaluations [20,48]. Failing to recognize these systemic connections frequently leads to treatment resistance, as the persistent internal drivers of inflammation remain unaddressed [3,5]. Integrating standard screening protocols, such as hydrogen and methane breath testing or targeted GI symptom questionnaires, allows dermatologists to catch these comorbidities early and build direct referral pathways with gastroenterologists [20,42]. This cooperative approach ensures that treatment protocols are synchronized, mitigating the risk of drug interactions while simultaneously addressing both the cutaneous and intestinal manifestations of the underlying disease process [11,15].

Final Remarks

Ultimately, the clinical management of refractory rosacea cases requires a transition toward a holistic, "gut-first" therapeutic approach [3,45]. When standard first-line topical and oral dermatological therapies fail to yield durable clinical remission, the underlying gastrointestinal microenvironment must be evaluated as the primary driver of the patient's persistent neurovascular and innate immune dysfunction [5,35]. Addressing localized intestinal dysbiosis, clearing small intestinal bacterial overgrowth with gut-directed antimicrobials like Rifaximin, and restoring mucosal barrier integrity offer a logical, root-cause path toward clearing systemic oxidative stress and the resulting skin flares [1,8,46]. Prioritizing the stabilization of the gut microbiome before or alongside traditional skin therapies not only achieves superior, long-lasting cutaneous clearance but also significantly enhances the patient's overall metabolic and systemic health [43,47]. As clinical evidence continues to solidify the mechanics of the gut-skin axis, moving toward a gut-first methodology transforms rosacea management from a practice of temporary symptom suppression into a strategy of true, systemic resolution [3,16].

Conclusion

The paradigm of rosacea management has fundamentally shifted from a narrow focus on topical symptom suppression to a systemic approach rooted in the gut-skin axis. It is increasingly clear that persistent, refractory facial flushing, papules, and pustules are frequently driven by underlying intestinal pathologies, particularly small intestinal bacterial overgrowth (SIBO) and profound colonic dysbiosis. The structural breakdown of the intestinal barrier permits a systemic spillover of bacterial antigens and pro-inflammatory cytokines, directly amplifying cutaneous innate immune cascades and neurovascular reactivity.

Validating this pathogenic bridge, targeted gastrointestinal stabilization—most notably through the eradication of SIBO using the gut-selective antibiotic rifaximin—yields high rates of durable, long-term dermatological remission. Moving forward, the successful management of complex rosacea phenotypes demands a multidisciplinary mandate. By combining conventional topical vasoconstrictors and vascular lasers with gut-directed antimicrobials, tailored probiotics, and anti-inflammatory

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dietary modifications, clinicians can transition toward a highly effective, "gut-first" treatment model. Ultimately, addressing the gastrointestinal microenvironment transforms rosacea therapy from a practice of temporary palliation into a strategy of permanent, root-cause resolution.

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