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Review Article

The Gut-Skin Axis: Exploring the Role of SCFAs, Obesity, and GLP-1 Receptor Agonists in Atopic Dermatitis

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Atopic dermatitis (AD), a chronic inflammatory skin disorder characterized by itching, erythema, and skin barrier dysfunction, has seen a global rise in prevalence alongside increasing rates of obesity. Emerging evidence highlights the gut-skin axis—a bidirectional communication network between gut microbiota and skin—as a critical link between these conditions. Central to this axis are short-chain fatty acids (SCFAs), microbial metabolites such as butyrate, propionate, and acetate, which exert anti-inflammatory effects and enhance skin barrier integrity. Obesity, associated with gut dysbiosis and reduced SCFA levels, exacerbates AD severity by impairing immune regulation and barrier function. This review explores the mechanistic links between obesity, SCFAs, and AD, emphasizing the role of the gut-skin axis. Additionally, it considers the potential role of glucagon-like peptide-1 (GLP-1) receptor agonists, conventionally used for type 2 diabetes and obesity, in AD pathogenesis. GLP-1 receptor agonists modulate gut microbiota, enhance SCFA production, and reduce systemic inflammation, potentially improving AD outcomes. While their direct application in AD remains underexplored, these agents represent an intriguing area for future investigation. Advancing this promising frontier in dermatology will require interdisciplinary collaboration and longitudinal studies to address gaps in understanding the gut-skin axis, SCFA mechanisms, and the potential of GLP-1 receptor agonists in AD.

INTRODUCTION

Atopic dermatitis (AD) is a chronic inflammatory skin disease with an increasing global prevalence that closely parallels rising obesity rates.[1-3] Clinically, it presents with pruritus, erythema, and compromised epidermal integrity. Pathogenesis involves a complex interplay between genetic predispositions—such as filaggrin gene (FLG) mutations—immune dysregulation favoring T-helper 2 (Th2) responses, and environmental triggers.[4-7]

Emerging research highlights the gut-skin axis, a bidirectional communication network linking gut microbiota to skin immune and barrier function, as a crucial modulator of AD.[8,9] Central to this axis are short-chain fatty acids (SCFAs), particularly butyrate, propionate, and acetate, which are produced via microbial fermentation of dietary fiber. SCFAs regulate immunity through histone deacetylase (HDAC) inhibition and support skin barrier repair via peroxisome proliferator-activated receptor gamma (PPAR-γ) and aryl hydrocarbon receptor (AhR) activation.[10-12]

Obesity further disrupts this axis by inducing gut dysbiosis. SCFA-producing taxa such as *Faecalibacterium prausnitzii* and *Roseburia* spp. are depleted, while pro-inflammatory lipopolysaccharide (LPS)-producing species like *Enterobacteriaceae* expand.[9,13,14] This microbial imbalance reduces SCFA availability and heightens systemic inflammation, thereby impairing immune regulation and epithelial barrier function—core elements in AD pathophysiology.[15,16]

Emerging evidence suggests that obesity-driven metabolic dysfunction may also alter cutaneous responses to microbial signals. For instance, Mendelian randomization studies confirm a causal relationship between elevated body mass index (BMI) and AD risk while molecular analyses reveal shared pathways involving adipokine dysregulation (eg, leptin resistance) and impaired PPAR-γ signaling.[17,18] These findings underscore obesity as a modifiable risk factor for AD severity.

This review explores how SCFA deficiency and gut microbial disruption mediate the link between obesity and AD. Furthermore, we propose a novel translational hypothesis: the repurposing of glucagon-like peptide-1 receptor agonists (GLP-1 RAs), commonly used in metabolic disease, as modulators of microbial composition, barrier integrity, and systemic inflammation in AD.[19,20] By integrating research across microbiology, immunology, and endocrinology, we aim to outline a roadmap for microbiota-centered AD therapies.

AD PATHOGENESIS AND THE MULTIFACETED ROLE OF SCFAS

AD pathogenesis is driven by an interdependent triad: immune dysregulation, epidermal barrier dysfunction, and

environmental factors.[5,6] Dysregulated Th2/Th17 immune responses promote cytokine release (eg, IL-4, IL-13, IL-17), leading to inflammation and epidermal disruption. Th2 cytokines suppress FLG expression, while Th17 activation encourages neutrophil infiltration. Regulatory T cells (Tregs), normally inhibitory, are functionally impaired in AD, resulting in sustained immune activation.[21-23]

Concurrently, barrier dysfunction stems from both inherited (eg, FLG mutations) and acquired defects. Impaired keratinocyte maturation, disrupted lipid metabolism, and weakened tight junctions reduce natural moisturizing factor (NMF) production and compromise barrier defenses.[4, 24-26] SCFAs play an integral role in restoring both immune and barrier balance. Through HDAC inhibition and free fatty acid receptor (FFAR2/3) signaling, they enhance Treg differentiation while suppressing Th2/Th17 polarization.[10, 20,23,27] SCFAs also inhibit TNF-α and IL-6 while increasing IL-10, fostering an anti-inflammatory environment.[28,29]

Cutaneously, SCFAs activate PPAR-γ and AhR pathways to stimulate keratinocyte differentiation and upregulate tight junction proteins such as claudin-1 and occludin.[7,11] They also mitigate oxidative stress and promote antimicrobial peptide production, strengthening the skin's defensive capacity.[18,26]

Clinically, diminished fecal and systemic SCFA levels are linked to increased AD severity.[2,8,29,30] Animal studies further confirm that butyrate or propionate supplementation restores immune balance and promotes barrier repair in AD models.[10,11] These findings position SCFAs as promising therapeutic mediators within the gut-skin axis and valuable targets for AD management.

OBESITY, GUT-SKIN AXIS DYSFUNCTION, AND AD PATHOGENESIS

The rising prevalence of AD has paralleled increasing obesity rates, prompting investigation into shared mechanistic pathways.[2,3] A key interface is the gut-skin axis, which integrates metabolic, immunologic, and microbial signaling. In obesity, chronic systemic inflammation exacerbates immune and barrier defects characteristic of AD. Longitudinal studies show that obesity more than doubles the incidence of AD and correlates with increased disease severity.[3,13,31]

Inflammatory cytokines such as TNF-α and IL-6 impair Treg function and intensify Th2/Th17 signaling, aggravating skin inflammation.[8,18,23] Concurrently, LPS translocation from the gut further disrupts epidermal structure by downregulating differentiation proteins and tight junctions.[25] Adipokine imbalances—namely leptin resistance and reduced adiponectin—suppress PPAR-γ signaling, enhancing oxidative stress and keratinocyte dysfunction.[18,32]

These processes are driven in part by obesity-associated gut dysbiosis. SCFA-producing taxa like *Faecalibacterium prausnitzii* and *Roseburia* spp. decline, while LPS-producing species expand.[9,13-16] This microbial shift reduces SCFA synthesis by up to 40%, with studies consistently showing lower fecal SCFA levels and microbial diversity in obese individuals compared to lean controls.[2,7,11,23,28-32] SCFA deficiency contributes to immune dysregulation, increased gut permeability, and impaired epidermal repair—all connected to AD pathogenesis.

Additionally, pro-inflammatory dietary patterns, such as those high in saturated fats, refined sugars, and processed foods, exacerbate both obesity and AD by driving chronic systemic inflammation and gut dysbiosis.[18,33] Conversely, anti-inflammatory dietary interventions including the Mediterranean diet, omega-3-rich foods, and increased fiber intake have demonstrated benefits in modulating microbiota composition, reducing cytokine levels, and improving skin barrier function.[34]

Together, these findings highlight how obesity-driven dysbiosis and SCFA depletion mechanistically link metabolic dysfunction to AD, positioning the gut-skin axis as a compelling therapeutic target.

GLP-1 RECEPTOR AGONISTS: A THEORETICAL LINK

Given these mechanisms, therapeutic strategies to restore SCFA balance and microbial integrity are beginning to emerge. Originally developed for the treatment of type 2 diabetes and obesity, GLP-1RAs have recently emerged as promising candidates for modulating the gut-skin axis in AD, most prominently in obese individuals with concurrent metabolic dysfunction.[35,36] Beyond their established metabolic effects, GLP-1RAs exhibit microbiome-modulating properties that could influence AD pathogenesis. Liraglutide, for example, has been shown to increase SCFA-producing bacteria such as *Lactobacillus* and *Bifidobacterium* by 2.1- to 3.4-fold, while suppressing inflammatory *Enterobacteriaceae* populations by over 60%.[19,37]

These microbial changes translate to improved intestinal barrier function: GLP-1RAs upregulate key tight junction proteins including occludin and ZO-1 by 45–80%, reducing LPS translocation by up to 73%.[19,38] This effect is mediated through GLP-1 receptor signaling on intestinal stem cells, promoting epithelial differentiation and barrier maintenance.[25] Downstream, these improvements in gut homeostasis may influence AD via three interconnected mechanisms. First, enhanced butyrate production increases filaggrin and loricrin expression while reducing transepidermal water loss (TEWL) and pro-inflammatory cytokine output.[10,11] Second, propionate stimulates endogenous GLP-1 secretion via FFAR2 activation on L-cells, creating a positive feedback loop that further amplifies gut-skin communication.[39,40] Third, GLP-1RAs themselves exert direct immunomodulatory effects, promoting Treg expansion and reducing Th17-mediated inflammation, with evidence of decreased serum IL-6 and TNF-α levels in treated patients (Please see Figure 1).[35,36]

While clinical data in AD remain limited, proof-of-concept exists in psoriasis: GLP-1 receptors are overexpressed in psoriatic plaques and GLP-1RAs modulate invariant natural killer T (iNKT) cells—key players in cutaneous inflammation.[41,42] These observations, coupled with evidence that GLP-1RAs improve keratinocyte barrier function via AMPK suggest broad applicability across inflammatory dermatoses.[43]

Early clinical data, such as semaglutide improving EASI-50 scores in 79% of AD patients, are promising, but robust trials are needed to validate GLP-1 RAs as AD therapies.[36] Ongoing studies (eg, NCT04869215) are investigating microbiome-targeted strategies, including prebiotic co-interventions, to optimize efficacy.[20] However, current evidence is largely confined to obese or metabolic syndrome populations, leaving their utility in non-obese AD patients unclear. Though still theoretical in dermatology, GLP-1 RAs represent a novel systems-level approach, concurrently addressing metabolic dysfunction, microbial dysbiosis, and immune dysregulation in AD.

RESEARCH GAPS AND FUTURE DIRECTIONS

Despite compelling evidence linking SCFAs, dysbiosis, and AD pathogenesis, several key research gaps limit the translation of these findings into clinical care. First, longitudinal studies examining gut microbiota composition and SCFA dynamics in obese individuals with AD are lacking, leaving the temporal sequence and causality of gut-skin interactions poorly defined.[13,31] Without such data, it remains unclear whether microbial shifts precede AD flares or simply co-occur.

Second, while preclinical models have demonstrated SCFA-mediated improvements in skin barrier function through PPAR-γ activation and HDAC inhibition, few studies have confirmed these pathways in humans—especially under conditions of obesity-associated inflammation, where metabolic and immunologic cross-talk may alter response dynamics.[11,23] The responsiveness of human keratinocytes and T cells to SCFA modulation in vivo remains an open question.

Most critically, neither SCFA supplementation nor GLP-1RAs have yet progressed to Phase III trials for AD treatment. This is surprising given the dual metabolic and immunologic benefits demonstrated in both mechanistic and early clinical studies.[36,40] Notably, the American Academy of Dermatology now recognizes obesity as a key comorbidity in AD yet guidelines lack specific recommendations for metabolic interventions.[44] Bridging this gap will require trials evaluating GLP-1RAs in AD patients with concurrent obesity, leveraging microbiome profiling to identify responders.

To close these gaps, future studies must adopt an interdisciplinary framework—integrating dermatologic, microbiologic, and metabolic endpoints—to evaluate treatment efficacy across systems. Priority interventions should include: (i) probiotic formulations targeting high-butyrate-producing taxa such as *F. prausnitzii*, (ii) combination regimens pairing GLP-1RAs with fermentable prebiotics, and (iii) SCFA-boosting dietary protocols tailored to individual microbiota profiles. Equally important is the development of non-invasive biomarkers to track gut-skin axis activity and personalize interventions based on patient-specific inflammatory and microbial signatures. These integrated approaches will be essential to translate microbiome science into meaningful, evidence-based therapies for patients with AD.

CONCLUSION

The convergence of microbiome science, immunology, and metabolic research has significantly advanced the understanding of AD, particularly in obese individuals where dysbiosis and SCFA depletion create a systemic environment conducive to chronic inflammation and barrier dysfunction.[9,30] Robust preclinical and translational evidence highlights the dual role of SCFAs in AD pathogenesis: as immune regulators—via HDAC inhibition and FFAR activation—and as structural enhancers of barrier function through PPAR-γ and AhR pathways.[10-12]

The emerging potential of GLP-1RAs as gut-skin axis modulators is a possible and exciting therapeutic frontier. As demonstrated in psoriasis, repurposing metabolic therapies for inflammatory skin diseases offers a paradigm shift. Targeting the gut–skin axis with GLP-1RAs may simultaneously address obesity-driven dysbiosis, SCFA depletion, and cutaneous inflammation, representing a precision medicine approach for refractory AD. These agents uniquely bridge microbial, metabolic, and immunologic domains, restoring SCFA-producing taxa, enhancing epithelial barrier integrity, and dampening inflammatory signaling.[19,20] Early clinical observations suggest meaningful skin improvements in patients receiving semaglutide, reinforcing the need for further investigation.[36]

Yet, critical questions remain. Future research would be beneficial to clarify how microbiome-targeted interventions, whether through diet, probiotics, or GLP-1RAs, impact skin outcomes and systemic inflammation in diverse AD populations. Interdisciplinary trials that integrate dermatologic, microbial, and metabolic endpoints will be key in identifying effective strategies and responsive subgroups.

Ultimately, the gut–skin axis offers more than a conceptual framework: it opens new therapeutic possibilities for one of the most burdensome chronic inflammatory skin diseases. As we move toward microbiome-informed care, restoring SCFA homeostasis may prove foundational in disrupting the cycle of obesity-driven AD and achieving durable clinical control.

DISCLOSURES

Dr. Lio reports being on the speaker's bureau for AbbVie, Arcutis, Eli Lilly, Galderma, Hyphens Pharma, Incyte, La Roche-Posay/L'Oréal, Pfizer, Pierre-Fabre Dermatologie, Regeneron/Sanofi Genzyme, Verrica; reports consulting/advisory boards for Alphyn Biologics (stock options), AbbVie, Almirall, Amyris, Arcutis, ASLAN, Bristol-Myers Squibb, Burt's Bees, Castle Biosciences, Codex Labs (stock options), Concerto Biosci (stock options), Dermavant, Eli Lilly, Galderma, Janssen, LEO Pharma, Lipidor, L'Oréal, Merck, Micreos, MyOR Diagnostics, Regeneron/Sanofi Genzyme, Sibel Health, Skinfix, Suneco Technologies (stock options), Theraplex, UCB, Unilever, Verdant Scientific (stock options), Verrica, Yobee Care (stock options). In addition, Dr. Lio has a patent pending for a Theraplex product with royalties paid and is a Board member and Scientific Advisory

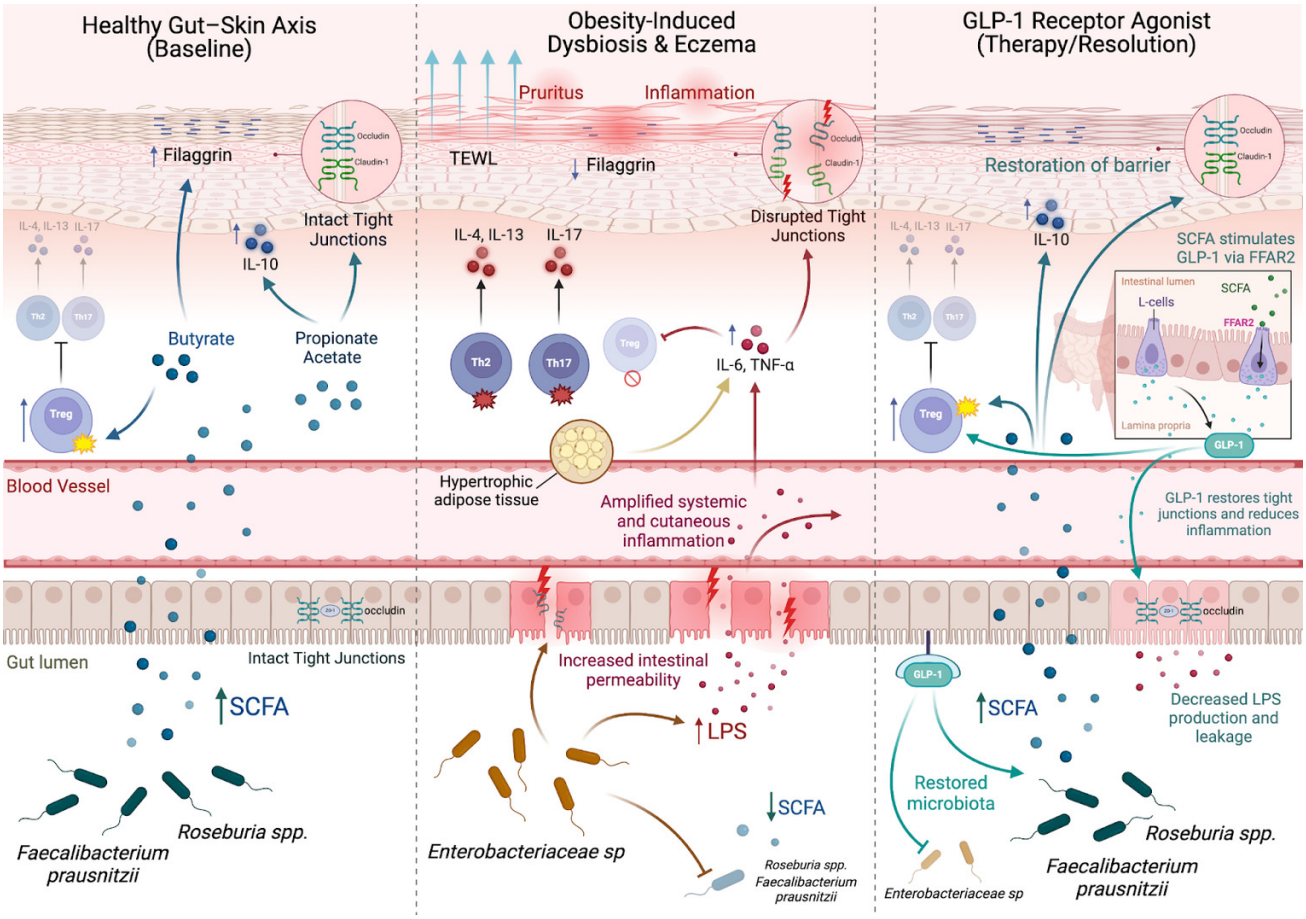

Figure 1. The gut–skin axis in atopic dermatitis (AD): obesity-induced disruption and potential restoration through GLP-1 receptor agonists.

This schematic illustrates the tripartite model of gut–skin axis regulation in AD: (1) **Healthy state (left panel):** SCFA-producing gut microbes (eg, *Faecalibacterium prausnitzii*, *Roseburia spp.*) maintain epithelial and immune homeostasis. Butyrate, propionate, and acetate enhance tight junction integrity (occludin, claudin-1), increase filaggrin expression, suppress Th2/Th17 polarization, and promote Treg differentiation via HDAC inhibition and FFAR2/3 signaling pathways.[10,11,23] (2) **Obesity-induced dysbiosis (center panel):** Expansion of *Enterobacteriaceae* and loss of SCFA-producing taxa reduce SCFA availability, increase intestinal permeability, and enable LPS translocation into systemic circulation, promoting chronic low-grade inflammation. This state impairs Treg activity, elevates Th2/Th17 cytokines (IL-4, IL-13, IL-17), and disrupts keratinocyte differentiation and skin barrier function.[9,15,18] Adipose tissue-derived cytokines (TNF-α, IL-6) further exacerbate cutaneous inflammation and oxidative stress. (3) **Therapeutic modulation by GLP-1 receptor agonists (right panel):** GLP-1RAs promote microbiota restoration (↑ *Roseburia*, *F. prausnitzii*), enhance SCFA levels, upregulate tight junctions, reduce systemic inflammation, and restore epidermal barrier integrity. SCFAs also stimulate endogenous GLP-1 secretion via FFAR2 on L-cells, creating a positive feedback loop.[19,20,39] Together, these pathways position GLP-1RAs as promising modulators of gut–skin axis dysfunction in AD.

Committee Member emeritus of the National AD Association.

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