World Journal of Gastrointestinal Pathophysiology

Submit a Manuscript: https://www.f6publishing.com

World J Gastrointest Pathophysiol 2025 September 22; 16(3): 108952

DOI: 10.4291/wjgp.v16.i3.108952

ISSN 2150-5330 (online)

REVIEW

Gut-skin axis: Emerging insights for gastroenterologists-a narrative review

Neeraj Singla, Khayati Singla, Mohamed Attauabi, Dhruv Aggarwal

Specialty type: Gastroenterology and hepatology

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification

Scientific Quality: Grade A, Grade B

Novelty: Grade B, Grade C

Creativity or Innovation: Grade B, Grade C

Scientific Significance: Grade A, Grade B

P-Reviewer: Al-Nimer MS, MD, PhD, Professor Emeritus, Iraq; Xu LQ, Chief Physician, China

Received: April 28, 2025

Revised: June 5, 2025

Accepted: August 25, 2025

Published online: September 22, 2025

Processing time: 145 Days and 1.8 Hours

Neeraj Singla, Medical Gastroenterology, Fortis Hospitals, Ludhiana 141123, Punjab, India

Khayati Singla, Department of Dermatology and Aesthetic sciences, Fortis Hospitals, Ludhiana 141123, Punjab, India

Mohamed Attauabi, Department of Gastroenterology and Hepatology, Copenhagen University Hospital-Herlev and Gentofte, Borgmester Ib Juuls Vej 1, DK-2730 Herlev, Denmark

Dhruv Aggarwal, Medical Student, Dayanand Medical College and Hospital, Ludhiana 141001, Punjab, India

Co-first authors: Neeraj Singla and Khayati Singla.

Corresponding author: Mohamed Attauabi, Department of Gastroenterology and Hepatology, Copenhagen University Hospital-Herlev and Gentofte, Borgmester Ib Juuls Vej 1, DK-2730 Herlev, Denmark. mohamed.attauabi.01@regionh.dk

Abstract

The gut-skin axis (GSA) embodies a complex, bidirectional interaction between the gastrointestinal (GI) system and skin, driven by immune modulation, systemic inflammation, and gut microbiota dynamics. Disruptions in gut homeostasis, including dysbiosis and increased intestinal permeability, are increasingly recognized as contributing factors to dermatological conditions such as acne, psoriasis, and atopic dermatitis. For gastroenterologists, appreciating this interplay is essential, as diseases and their treatments frequently present with cutaneous manifestations, offering diagnostic and therapeutic insights. This review explores the underlying mechanisms of the GSA, focusing on the microbiome and its metabolites as key regulators of inflammation and immunity. It underscores the clinical importance of microbiome-targeted therapies, such as probiotics, prebiotics, and dietary modifications, in addressing both GI and dermatological disorders. Furthermore, the review examines the influence of GI conditions, including inflammatory bowel disease and celiac disease on skin health. This article seeks to equip gastroenterologists with practical insights for identifying, diagnosing, and managing skin conditions associated with GI health. The article also highlights the current limitations in knowledge regarding the GSA. The GSA represents a promising avenue for therapeutic advancements, encouraging interdisciplinary collaboration between gastroenterology and dermatology to optimize patient care.

Key Words: Gut-skin axis; Microbiome; Inflammatory bowel disease; Celiac disease; Psoriasis; Dysbiosis; Atopic dermatitis; Probiotics; Faecal microbiota transplant

Core Tip: The gut-skin axis reflects a robust bidirectional connection between the gut and skin, driven by immune and metabolic pathways. Intestinal dysbiosis is a key driver of systemic inflammation, impacting both the gut and skin. Dietary influences, atopy, and microbial modulation play critical roles in this interplay. Skin disorders like atopic dermatitis (AD), hidradenitis suppurativa, psoriasis have association with gastrointestinal (GI) diseases. They might also serve as an early marker of underlying GI pathology. Microbiome-targeted therapies like probiotics and fecal microbiota transplantation offer promising avenues in the management of skin disorders like psoriasis, AD and acne rosacea rooted in gut dysregulation.

Citation: Singla N, Singla K, Attauabi M, Aggarwal D. Gut-skin axis: Emerging insights for gastroenterologists-a narrative review. *World J Gastrointest Pathophysiol* 2025; 16(3): 108952
URL: https://www.wjgnet.com/2150-5330/full/v16/i3/108952.htm
DOI: https://dx.doi.org/10.4291/wjgp.v16.i3.108952

INTRODUCTION

Numerous species can be found in the human gut and skin, with the gut harboring 10^{14} microbial cells and the skin homing 10^{12} microbial cells[1,2]. The microbiome contributes significantly to host health by modulating the immune system, protecting against pathogens, aiding in metabolite digestion, and maintaining barrier function. The development of next-generation sequencing over the last decade has completely changed our knowledge of the composition of the microbiome and provided previously unheard-of insights into its presence in the gut and on the skin[3,4]. Within the larger gut-brain-skin-immune axis, the gut-skin axis (GSA) can be viewed as an essential component. Recent research highlights the crucial role that gut dysbiosis plays in the emergence of numerous inflammatory disorders by pointing to a reciprocal relationship between altered gut microbiome and skin homeostasis abnormalities through immune system alteration[5,6].

This review will provide an organized perspective on the intricate relationship between the gut and skin microbiomes and their relevance to gastrointestinal (GI) disorders such celiac disease and inflammatory bowel disease (IBD). The article's secondary objective is to explore dermatological manifestations associated with GI disorders.

UNDERSTANDING THE GSA

The gut and skin function as important contact organs, serving as the means through which the mammalian body interacts with its surroundings. Emerging evidence indicates that the skin, which serves as a physical barrier against the external environment and is a highly vascularized organ that is metabolically and immunologically active, possesses its own unique microbiome and can influence gut health[7]. The GSA signifies a strong bidirectional link between the gut and skin, where immune and metabolic processes enable mutual influence on their health[8,9]. Indeed, it has been proposed that the axis is a component of a more extensive interconnected network, the gut-immune-brain-skin axis, which entails complex communication between gut microbiota, immune and central nervous systems, and the skin, as noted by Vojvodic *et al*[10] in 2019 (Figure 1).

According to the national institute of health (Human Microbiome Project), for instance, the GI tract and skin contain roughly 50% of the entire bacterial population (29% in the GI tract and 21% in the skin, respectively)[11]. While the skin's surface area averages around 2 m^2, the inclusion of various skin appendages and openings—such as sebaceous glands, eccrine and apocrine ducts, and hair follicles—expands the total epithelial interface for microbial colonization to over 25 m^2[3]. The gut microbiota, often referred to as the "virtual organ", plays a crucial role in immune regulation and metabolism, influencing not only the skin but also other organs such as the lungs and brain[12].

The precise mechanism that explains the interactions between gut and skin microbes has not been completely clarified yet, but it is likely that the immune and endocrine systems play a role in this process[13]. The most widely accepted theory amongst multiple theories is that intestinal dysbiosis may be the root cause of inflammation in both the gut and skin, ultimately leading to the GSA's detrimental effects on the skin[14]. The metabolic waste products of gut bacteria may also get into the bloodstream and build up in the skin by direct migration or due to increased intestinal permeability, which could reduce keratin production and hence affect epidermal differentiation at the same time[15,16].

In fact, the influence of microorganisms and metabolites on gut permeability and inflammatory response—both of which are variable—is directly linked to their effect on skin aging. Additionally, emerging evidence suggests that resident cutaneous commensals may exert additional immunomodulatory effects, potentially influencing systemic inflammatory responses. Accordingly, dysbiosis of the cutaneous microbiome, often observed alongside gut dysbiosis, has been linked

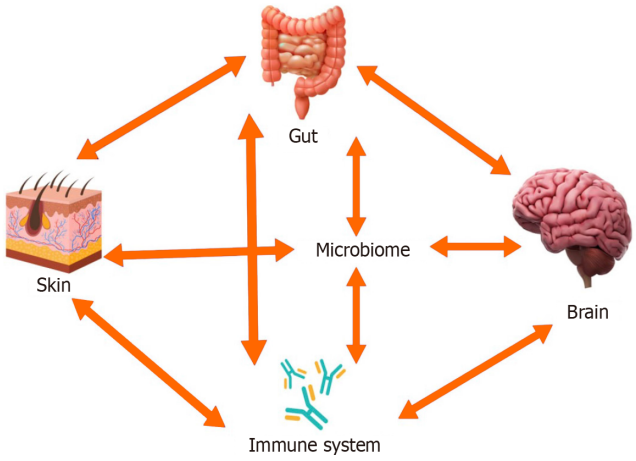

Figure 1 Gut-skin axis as part of the broader skin-gut-brain-immune system axis. The skin, gut, brain, and immune system are intricately interconnected, engaging in a dynamic and bidirectional communication network. This cross-talk includes skin–gut, skin–immune, gut–brain, and gut–immune interactions. At the heart of this complex axis lies the microbiome, orchestrating the interplay across these systems and playing a pivotal role in health and disease.

to the development of inflammatory skin conditions such as atopic dermatitis (AD), rosacea, and psoriasis[15]. Whether targeted modulation of the gut microbiota can exert reciprocal effects on cutaneous microbial communities remains insufficiently understood.

The neuroendocrine connection between the gut and skin microbiota is driven by the ability of gut microbes to stimulate brain circuits through the production of neurotransmitters such as noradrenaline, serotonin, and acetylcholine [17,18]. The zones of referred pain, also known as Head's zones, are another component of the gut-brain-skin axis. These are the areas of skin where pain that originates in different visceral organs projects itself[18].

The emergence of atopy is yet another connection between the gut and skin[19]. Although the majority of the data suggests that the gut microbiota affects the skin, the opposite may also be true. Allergic reactions can be seen in the first few months of infancy in the case of type I food-specific IgE-mediated responses, indicating that sensitization happens before food ingestion by exposure through inflammatory skin rather than the GI tract[20]. The theory suggests that oral tolerance is bypassed when allergens like peanuts, wheat, and eggs are first introduced through the skin. The significance of food in the gut skin axis is demonstrated by the close correlation between AD and food allergies[21].

Understanding the GSA requires taking into account the ethnic variations in dietary habits and, consequently, genetic impacts. For example, populations adhering to traditional hunter-gatherer lifestyles have a reduced incidence of acne, whereas individuals who consume a high-fibre Mediterranean diet have a decreased risk of AD[22].

In a clinical context, GI and skin symptoms linked to conditions like rosacea, celiac disease, and IBD indicate the presence of the GSA. Although the association is thought to be reciprocal, dysbiosis and the resulting inflammation in the gut have been identified as the primary trigger in the majority of cases[8].

MECHANISM OF INTERACTION

To better understand how the GSA operates at the molecular level, this section highlights key pathways linking microbial metabolites to immune and barrier responses relevant to skin health. Gut dysbiosis alters the production of microbial metabolites, which in turn activate proinflammatory pathways *via* epithelial and immune cell signalling. The interaction between gut microbiota and intestinal epithelial cells can be facilitated by short-chain fatty acids, secondary bile acids (including deoxycholic acid, lithocholic acid, and ursodeoxycholic acid), and tryptophan-derived metabolites from gut microbiota (such as indole and its derivatives)[23] (Figure 2).

A multitude of biological and environmental variables, including chronic stress, antibiotic usage, exposure to environmental pollutants, and a pro-inflammatory diet, lead to gut dysbiosis and therefore 'leaky gut'. Leaky gut syndrome may explain the increasing evidence of the association of intestinal disorders with autoimmune conditions and inflammatory skin conditions such as acne vulgaris, psoriasis, and chronic urticaria[24].

Specific metabolites, such as free phenol and p-cresol, are generated by gut bacteria, particularly *Clostridium difficile*. P-cresol is a biomarker indicative of GI disturbance. *In vitro* evidence indicates that p-cresol and phenol diminish the expression of keratin type 10 in cultured keratinocytes, potentially affecting epidermal development and barrier function. Moreover, research in humans indicates that the restriction of probiotics leads to increased cresol concentrations in the blood, along with diminished skin moisture and a reduction in corneocyte size[25] (Table 1).

In several murine investigations, probiotic-fed animals displayed notable skin alterations which subsequently stimulated the generation of T regulatory lymphocytes. Consequently, T regulatory cells possess the capacity to influence the immune system outside the GI tract[8].

A key aspect of GSA is atopy development. Loss-of-function mutations in filaggrin, a protein crucial for the epidermal barrier, are associated with peanut allergy. Peanut allergens persist in dust and can be encountered through the skin

Table 1 Key microbial metabolites and their effects on the gut-skin axis

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)

SCFA: Short-chain fatty acid.

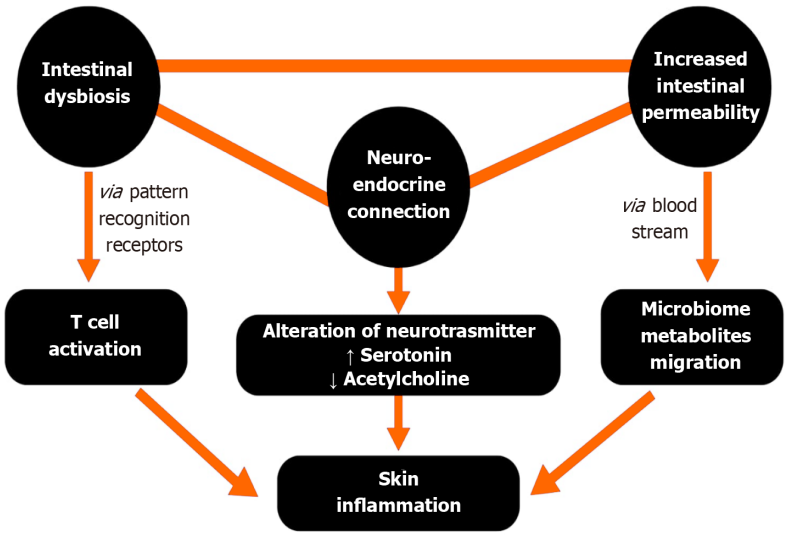

Figure 2 Mechanism of gut-skin axis. Intestinal dysbiosis, increased gut permeability, and the neuro-endocrine axis are the three central mechanisms linking the gut and skin. Dysbiosis activates T cells through pattern recognition receptors, while increased intestinal permeability facilitates the translocation of microbial metabolites. Additionally, neurotransmitter imbalances—characterized by elevated serotonin and reduced acetylcholine—further contribute to the disruption. These combined factors—T cell activation, altered neurotransmission, and circulating microbial metabolites—ultimately drive skin inflammation.

before GI exposure. Studies suggest that early oral exposure to peanuts (before 12 months) lowers allergy risk in high-risk individuals, highlighting the importance of oral exposure before skin contact to prevent allergy development[21]. Patients with AD exhibit diminished barrier function, both in the epidermis and the intestinal mucosa facilitating the recurrent transmission of foreign antigens resulting in the onset of symptomatic AD[22].

Beyond microbial metabolites, dietary components also play a pivotal role in modulating the GSA, particularly in conditions such as acne where nutrient-driven metabolic signalling influences inflammation and microbiota composition. A high intake of carbohydrates and saturated fats disrupts nutritional signalling, affecting key regulators like forkhead box O1 and mammalian target of rapamycin complex 1 (mTORC1), which control lipogenesis *via* sterol regulatory element binding protein (SREBP-1). In acne patients, excessive SREBP-1 activation increases monounsaturated fatty acid production, promoting *Propionibacterium acnes* growth and IL-1 secretion, leading to comedogenesis. A low-glycaemic diet with metformin inhibits mTORC1, making it a promising treatment for acne, hidradenitis suppurativa, and rosacea [26].

Another crucial component of GSA is its connection to the neuroendocrine system. Gut microbiota changes can induce systemic inflammation, activating microglia and contributing to depression. Cytokines like IL-6 stimulate the hypothalamic-pituitary-adrenal axis, increasing cortisol production and altering tryptophan metabolism, leading to reduced serotonin synthesis[27,28]. The skin functions as a neuro-immune-endocrine organ, generating and reacting to hormones, cytokines, and neurotransmitters, including neuropeptides. The hair follicle is regarded as the most metabolically active skin appendage, involved in aerobic glycolysis and epithelial mitochondrial metabolism that governs

neuroendocrine regulation[29] (Table 2).

CLINICAL IMPLICATION FOR GASTROENTEROLOGISTS

Given the role of gut-skin interactions in systemic inflammatory states, it is not surprising that several GI diseases, particularly IBD, have well-documented dermatological associations. The GSA has role in both pathological and functional gut disorders including irritable bowel syndrome (IBS). This section categorizes and describes these manifestations. It is evident that certain overlaps between gut and skin diseases may be attributable to genetic factors (*e.g.*, certain polyposis syndromes and hereditary GI malignancies or to common pathobiological mechanisms (*e.g.*, systemic vasculitis) (Table 3).

IBD and associated skin conditions

Cutaneous manifestations linked to IBD can be categorized into three groups based on their pathological characteristics: Those exhibiting features analogous to the affected GI tract (specific lesions), those that do not (reactive lesions), and associated lesions likely related to human leucocyte antigen linkage and the chronic inflammatory nature of IBD.

Specific lesions result from the direct involvement of the skin by the identical pathogenic process observed in the GI tract. These manifestations are exclusively observed in Crohn's disease (CD), while ulcerative colitis (UC) often does not affect mucosal membranes. These encompass: (1) Perianal fissures and fistulas which occur in 36% of people with CD; and (2) Oral CD: Oral lesions manifest in 5%-20% of individuals with CD. The lesions do not consistently correspond with the activity of the underlying intestinal pathology. It can present as aphthous stomatitis, pyostomatitis vegetans (PSV), angular cheilitis, ulceration, mucosal nodularity, nodules of gingival and alveolar mucosa, and indurated fissuring of the lower lips, frequently painful and obstructive to consumption. Oral lesions are more common with CD, and vice versa. PSV is the sole oral lesion that manifests more frequently in UC[30].

Metastatic CD

Distinct granulomatous cutaneous lesions exhibiting identical histology to the intestinal lesions. Metastatic CD is primarily seen on the extremities or in intertriginous regions, with face and genital lesions infrequently documented.

Reactive lesions are observed in both UC and CD. Cross antigenicity between the dermis and intestinal mucosa may account for reactive lesions. These include erythema nodosum (EN), pyoderma gangrenosum (PG), PSV and pyodermatitis vegetans (PDV).

EN: EN is the most common skin symptom of IBD, affecting 3%–10% of UC and 4%–15% of CD patients, with a higher prevalence in women and HLA-B27 carriers[31]. It typically appears about five years after UC diagnosis and rarely precedes it. In CD, EN is more commonly seen with colonic involvement[32]. EN often coincides with UC flares but doesn't always reflect disease severity. It also occurs in infectious, non-infectious, and idiopathic conditions. EN results from immune complex deposition, triggering a Type IV hypersensitivity reaction. Clinically, it presents as sudden, symmetrical, erythematous, deep nodules (1–5 cm) on the lower limb extensor surfaces but can appear elsewhere[33-35].

PG: PG is the second most common skin condition linked to IBD after EN. It is a painful, non-infectious neutrophilic dermatosis that rapidly progresses from erythematous lesions to necrotic ulcers. Up to 50% of cases have underlying systemic diseases, with IBD involved in 30%[36]. Clinically, PG presents with jagged, undermined edges and a violaceous border, mainly affecting the lower legs but also the genitals, oral mucosa, and rarely, internal organs.

Peristomal PG occurs in 0.6% of stoma patients yearly, often triggered by minor irritation, leading to painful ulcers that impair stoma function and social well-being. Postsurgical PG, linked to IBD in 5.9% of cases, starts as wound dehiscence in a normal scar. PG can appear before, during, or after IBD onset, independent of disease activity, significantly impacting patient morbidity and quality of life[37].

PSV and PDV: PSV is a rare oral condition with pustules on the mucosa that rupture into fissured "snail-track" lesions, mainly affecting the gingiva, palate, and buccal mucosa. More common in young to middle-aged males, it often follows IBD, necessitating GI evaluation if no prior diagnosis exists. Treating IBD improves symptoms[38,39].

PDV, an uncommon IBD-related skin condition, presents as pustules and ulcerations in folds like the axilla or groin but can also affect the face, scalp, trunk, or limbs. Pustules rupture into erosions with hemorrhagic bases and distinct vegetative plaques[39].

Associated lesions

These include aphthous stomatitis, psoriasis, vitiligo, hidradenitis suppurativa, reactive arthritis, eczema, clubbing of the nails, and acrodermatitis enteropathica.

Aphthous stomatitis

Up to 10% of IBD patients develop this third most common extra-intestinal symptom, marked by painful, round or oval ulcers on the buccal or labial mucosa[40,41]. Oral lesions often coincide with IBD flare-ups and are linked to iron, folic acid, and B12 deficiencies. Histology suggests a neutrophilic dermatosis, possibly influenced by *Streptococcus sanguinis* [33]. *Acinetobacter johnsonii* is found in recurrent cases, while healthy oral micobiota especially *Streptococcus salivarius* decreases[42].

Table 2 Shared cytokine pathways in gut and skin disorders

Cytokine	Source	Role in gut disease	Role in skin disease
IL-17	Th17 cells	Promotes mucosal inflammation in Crohn's disease; may have protective roles in UC	Drives psoriasis, AD
IL-23	Dendritic cells, macrophages	Supports Th17 expansion in IBD	Involved in psoriatic inflammation
TNF-α	Macrophages, monocytes	Central to both Crohn's and UC pathogenesis	Targeted in psoriasis, hidradenitis suppurativa
IL-4	Th2 cells	Mucosal immunity shift in some IBD variants	Hallmark of atopic dermatitis
IL-6	Multiple immune cells	Acute phase reactant in IBD	Links gut-brain-skin inflammation
IFN-γ	Th1 cells	Epithelial barrier disruption	Found in psoriatic and alopecia lesions
IL-22	Th17 cells, innate lymphoid cells	Promotes epithelial regeneration, but may drive pathology in IBD (*e.g.*, UC)	Promotes keratinocyte proliferation in psoriasis

IL: Interleukin; UC: Ulcerative colitis; IBD: Inflammatory bowel disease; AD: Atopic dermatitis; IFN-γ: Interferon-gamma.

Table 3 Common diseases/syndromes with their gastrointestinal and skin manifestations

Disease/syndrome	Skin manifestations	Associated GI malignancy	GI and other features
Muir Torre syndrome (Variant of Lynch syndrome)	Sebaceous adenoma. Epitheliomas. Multiple keratocanthomas	Colon, gastric, hepato-biliary, pancreas	Cancer involving endometrium, cervix, lung, urological, blood, ovary
Gardner syndrome	Epidermoid cysts. Lipomas. Desmoid tumors	Colorectal, duodenal, hepatoblastoma, stomach	CHRPE, supernumerary teeth, adrenal tumors, osteomas
Peutz Jegher's syndrome	Hyperpigmentation of tongue. Pigmentation around lips around mouth	Duodenal, colon, pancreas, stomach, small bowel	Cancer involving pancreas, breast, ovary, genitals
Cowden syndrome	Trichilemmomas. Oral papillomatosis. Facial papules. Acral keratoses	Colon	Melanoma, cancer involving endometrium, thyroid, kidney
Bannayan-Riley-Ruvalcaba syndrome	Hyperpigmented macules involving Glans penis or vulva. Acanthosis nigricans. Facial verrucous papules	None	Macrocephaly, hypotonia, autism spectrum disorder, development delay, hamartomas, seizures, scoliosis
Juvenile polyposis syndrome	Digital clubbing. Facial and digital telangiectasias	Colon, SI, stomach	Cancer involving. Pancreas
Cronkhite Canada syndrome	Nail dystrophy. Alopecia. Hyperpigmentation	Gastric, colon	Intestinal mucosal changes leading to malabsorption
Bazex syndrome (Acroker-atosis Paraneoplastica)	Scaly. Psoriasiform plaques on acral areas, and nasal, ear and malar surfaces	Upper GI tract	SCC of pharynx, esophagus, larynx, lung, lymphoma and genito-urinary tumors
Tylosis	Palmoplantar hyperkeratosis	Esophagus (SCC)	
Plummer Vinson syndrome	Koilonychia	Esophagus (SCC)	Esophageal web, iron deficiency
Glucagonoma and Necrolytic Migratory erythema	Annular erythematous eruption with blisters. Ngular cheilitis. Glossitis. Stomatitis		Anemia, weight loss, diarrhoea, steatorrhoea, thromboembolic disease, psychiatry disturbance
Carcinoid syndrome	Flushing. Rosacea. Pellagra like changes. Cutaneous metastasis	Appendix, small intestine	Wheezing, carcinoid heart disease, diarrhoea
Paraneoplastic dermatomy-ositis	Heliotropic rash. Gottron papules. Violaceous poikiloderma. Ragged cuticle. Nail fold telangiectasias	Stoamch, colorectal, pancreas	Dermatomyositis, cancer involving lung and ovary, lymphomas
Cutaneous metastasis	Sister Joseph Mary nodule on umbilicus	Gastric adenocarcinoma	

GI: Gastrointestinal; SCC: Squamous cell carcinoma; SI: Small intestine; CHRPE: Congenital hypertrophy of the retinal pigment epithelium.

Psoriasis

IBD and psoriasis are chronic inflammatory diseases with relapsing-remitting courses, affecting the gut and skin barriers, respectively. Both peak between ages 15-30 and share immune dysregulation driven by genetic and environmental factors. Psoriasis affects 3%-4% of IBD patients, more commonly in CD, with shared susceptibility genes like interleukin (IL)-23R and overlapping inflammatory pathways involving tumor necrosis factor-alpha, IL-23, IL-17, and IL-22[43].

Despite IL-17A's role in both diseases, its inhibition improves psoriasis but worsens CD. Gut dysbiosis in psoriasis may trigger autoimmune responses through endotoxin-peptidoglycan superantigens. Some biologics, like infliximab, can induce secondary inflammatory diseases, such as psoriasis in CD patients, due to cytokine imbalances - a phenomenon referred to as paradoxical inflammation[44]. Additionally, microscopic gut inflammation is common in all spondyloarthritis subtypes, indicating intestinal barrier dysfunction[45].

Celiac disease and its associated skin conditions

Dermatitis herpetiformis (DH) is the key skin disorder linked to celiac disease and serves as a diagnostic marker. It is an autoimmune condition with intensely itchy, symmetrical lesions on the extensor surfaces, including the elbows, knees, scalp, and buttocks. Lesions vary from erythema and urticarial plaques to papules, blisters, erosions, and hyperpigmentation[45]. DH presents similarly in adults and children, though children may show atypical features like hemorrhagic palmoplantar lesions, deep nodules, and facial involvement[46].

Diagnosis involves clinical evaluation, histopathology, immunofluorescence, and serology. Direct immunofluorescence of perilesional skin is the gold standard, while IgA anti- tissue transglutaminase and anti-endomysial antibody antibodies help detect gluten sensitivity. A gluten-free diet is the main treatment, with dapsone, sulfasalazine, potent topical corticosteroids, and antihistamines as alternatives for symptom relief[47].

Additional dermatological conditions linked to celiac disease encompass diseases demonstrated to improve with a gluten-free diet or the presence of serological markers of celiac disease include psoriasis, alopecia areata, urticaria, cutaneous vasculitis, AD, and chronic ulcerative stomatitis, among others.

Diseases resulting from dietary deficiencies: Perioral and perianal dermatitis, alopecia, genital lesions (zinc deficiency), alopecia, xerosis, pruritus, koilonychia (iron deficiency), angular stomatitis (iron and vitamin B12 deficit), spiny skin (vitamin A deficiency), pellagra (niacin/vitamin PP insufficiency: Pellagra prevention). Dental complications: Enamel anomalies, delayed eruption.

IBS and acne rosacea

There is growing evidence to indicate that acne rosacea tends to occur more commonly in individuals suffering from IBS. This association between rosacea and GI conditions is believed to be mediated by shared genetic, microbial, and immune-related factors, forming the basis of the GSA. Persistent exposure to inflammatory triggers—such as certain foods, alcohol, and psychological stress—not only worsens rosacea but may also contribute to a state of chronic inflammation. This sustained inflammatory response might activate similar immune pathways implicated in IBS as well. Moreover, neurovascular alterations seen in rosacea could influence gut motility and visceral sensitivity, thereby playing a role in the manifestation of IBS symptoms[48].

MICROBIOME AND SKIN DISORDERS

The GSA reflects the bidirectional connection between gut and skin microbiomes and is supported by several observations as discussed in the next sections. From a clinical perspective, AD is associated with increased risk of IBD, cutaneous para-neoplasia is associated with increased risk of GI malignancies, psoriasis is associated with increased risks of UC and celiac disease, and hidradenitis suppurativa is associated with elevated risks of CD and UC[49].

AD

Epidermal barrier dysfunction is central to AD pathogenesis. Flaring lesions often show *Staphylococcus aureus* overgrowth, correlating with disease severity. *Staphylococcus aureus* produces proteases and phenol-soluble modulin α, which compromise the skin barrier, induce inflammation, and promote biofilm formation[50].

Recent evidence implicates gut microbial composition as a key modulator of skin inflammation, underscoring its role in the pathogenesis of AD and related conditions. Reduced *Faecalibacterium prausnitzii* disrupts gut barrier integrity and promotes Th2 responses typical of AD. Greater gut microbial diversity in infants is associated with lower AD risk[51-53].

AD patients frequently exhibit decreased *Bifidobacterium* and *Bacteroides*, with increased *Enterobacteriaceae*. Fungal shifts include elevated *M. osloensis* and *M. luteus*.

Diet also influences AD, with lower intake of antioxidants and omega-3 fatty acids, and higher omega-6 fatty acid consumption, linked to increased disease susceptibility[54].

Psoriasis

Psoriasis flare-ups can be triggered by microbes such as *Staphylococcus aureus*, *Streptococcus pyogenes*, *Candida albicans*, *Malassezia*, and viruses like human papilloma virus and endogenous retroviruses. Psoriatic plaques often show *Corynebacterium*, *Propionibacterium*, *Staphylococcus*, and *Streptococcus* species[55,56].

Both skin and gut dysbiosis influence psoriasis onset and flares. Gut microbiome changes include reduced *Akkermansia muciniphila* and increased *Ruminococcus*, *megasphaera*, and other firmicutes and actinobacteria (*e.g.*, *R. gnavus*, *D.*

formicigenerans, *C. aerofaciens*), with lower *Prevotella copri* and *Parabacteroides distasonis*. Bacterial translocation (*e.g.*, *Escherichia coli* deoxyribonucleic acid) has been linked to systemic inflammatory markers and earlier disease onset[57,58].

Rosacea

Rosacea is associated with distinct microbial patterns. *Demodex folliculorum* density is up to 10 times higher in rosacea lesions. *Gordonia* and *geobacillus* species correlate with rosacea society scores. In papulopustular rosacea, *Campylobacter ureolyticus* and *Prevotella intermedia* are elevated, while *Acinetobacter* and *Cutibacterium* are depleted[59,60].

Microbiome shifts also differ by subtype: *Firmicutes* and *Actinobacteria* are more abundant in erythematotelangiectatic and papulopustular rosacea, respectively. Additionally, *Prevotella copri* may be reduced, whereas *Bacteroidales*, *Syntrophomonadaceae*, *Lachnospiraceae*, *Anaerovorax*, *Tyzzerella*, *Akkermansia muciniphila*, and *Parabacteroides distasonis* are increased compared to healthy controls[61].

Hidradenitis suppurativa

Hidradenitis suppurativa is a rare immune-mediated chronic skin disease affecting 0.3% of the global population and primarily manifesting as occlusion of the apocrine glands. Lesions exhibit increased levels of anaerobic species, *e.g.*, *Prevotella* and *Porphyromonas, Fusobacterium*, and *Parvimonas* spp. correlating to disease severity. A pilot study reported reduced skin microbial diversity, coupled with elevated *Bilophila* and lower *Lachnobacterium* in hidradenitis lesions. Reduced alpha diversity has been noted in both faecal and skin samples from patients with hidradenitis[62,63].

ROLE OF PRO/PRE-BIOTICS IN GSA

The concept of using gut microbiome therapies to influence inflammatory skin diseases has been explored for some time. In a seminal paper, it was demonstrated that in a mouse model with psoriasis induced by imiquimod, the probiotic strain *Lactiplantibacillus pentosus* GMNL-77 treated mice experienced significantly less erythema, scaling, and epidermal thickening compared to untreated control mice, by modulating inflammation-associated cytokines tumor necrosis factor-α, IL-6, IL-23, and IL-17A[64] (Table 4).

In psoriasis, it was demonstrated in animal studies that *Bifidobacterium longum* (*B. longum*) subsp *infantis* 35624 induces regulatory T cells that function beyond the gut environment[65]. Consistent with these findings, *Bifidobacteria infantis* 35624 supplementation in patients with psoriasis reduced circulating C-reactive protein and tumor necrosis factor-α levels [66]. Another study in plaque psoriasis investigated a multistrain probiotic (incorporating *Lactobacillus acidophilus, Bifidobacterium bifidum, B. lactis, and B. longum*) and found improvements in depression (Beck's Depression Inventory), quality of life (Dermatology Life Quality Index), and disease severity [Psoriasis Area and Severity Index (PASI)], coupled with elevated total antioxidant capacity and reduced C-reactive protein and IL-6[67]. Finally, *Bacteroides fragilis* BF839 has shown promise in a preliminary open-label trial, reducing PASI scores in psoriasis[68].

The frequent coexistence of GI and dermatologic pathology suggests a therapeutic opportunity, *e.g.*, targeting the gut microbiome to manage systemic inflammation. The following sections outline microbiome-directed strategies currently being explored for their effects on skin conditions.

Gut dysbiosis has been noted in disorders including AD and rosacea, where the elimination of the related small intestine bacterial overgrowth results in substantial regression of the skin lesions[69]. Likewise, certain metabolites such as curcumin, lycopene, polyphenols, and dietary rice prolamine extracts have been shown to enhance the skin barrier. Recent data indicates that the modification of gut microbiota through the consumption of prebiotics, probiotics, postbiotics, and/or synbiotics can significantly affect skin health and the manifestations of skin aging[70].

In pediatric patients with AD, supplementation with a probiotic blend consisting of *Bifidobacterium animalis subsp. lactis CECT 8145, B. longum CECT 7347, and lacticaseibacillus casei CECT 9104* (formerly *Lactobacillus casei CECT 9104*) significantly improved scoring for AD indices[71]. Interestingly, a meta-analysis of 22 clinical trials investigating the use of prebiotics for allergy prevention demonstrated a significant reduction in the incidence of AD[72].

Probiotic strains must remain viable in sufficient concentrations at the time of ingestion to exert clinical effects. Emerging evidence suggests that long-term dietary patterns may exert broader effects on gut microbial composition compared to short-term supplementation with probiotics or prebiotics, as both dietary fibre and weight reduction can diminish the prevalence of inflammatory bacteria. Certain probiotic and prebiotic interventions have been shown to modulate regulatory T cell responses and may support the restoration of barrier integrity in experimental models. Prebiotics, similar to probiotics, can markedly reduce the production of harmful fermentation byproducts, which may be advantageous for patients with AD[73].

The World Allergy Organization does not endorse the use of prebiotics during pregnancy or breastfeeding as a preventive strategy for AD due to insufficient scientific data. Although other meta-analyses have indicated a marginally advantageous role due to immunological training in-utero[74]. A meta-analysis conducted by Pelucchi *et al*[75] indicated a small effect of dietary supplementation with probiotics during pregnancy in preventing AD in infants.

Systemic probiotics may potentially reduce inflammation in acne. Probiotics decrease sebum levels, which leads to lesser follicular colonization by *P acnes* and consequently decreased inflammation. These trials were predominantly short-term, highlighting the necessity for research on the long-term effects of lactobacillus extract in acne lesions[73].

Role of ultraviolet B light and vitamin D on gut microbiome

Skin exposure to ultraviolet B light, and the resulting rise in serum vitamin D levels, has been shown to enhance both the α- and β-diversity of the gut microbiome. This exposure led to the enrichment of several bacterial families, with serum

Table 4 Gut-skin axis–linked diseases and their microbial dysbiosis profiles

Condition	Gut microbiome changes	Skin microbiome changes
Atopic dermatitis	↓*Bifidobacteria*, ↓*F. prausnitzii*, ↑*Enterobacteriaceae*	↑*S. aureus*, ↓microbial diversity
Psoriasis	↑*Ruminococcus*, ↓*Akkermansia muciniphila*, ↓*Faecalibacterium prausnitzii*	↑*Corynebacterium*, ↑*Streptococcus*
Rosacea	↑*Prevotella intermedia*, ↓*Cutibacterium*	↑*Demodex folliculorum*, ↑*Gordonia*
Hidradenitis suppurativa	↑*Porphyromonas*, ↑*Prevotella*, ↓diversity	↑*anaerobes* in lesions, ↓alpha diversity
Celiac disease	↑*Proteobacteria*, ↓*Lactobacillus*, ↑*Candida*	Associated with dermatitis herpetiformis
Seborrheic dermatitis	Altered lipid metabolism-related bacteria (*e.g.*, ↑Enterobacteriaceae)	↑*Malassezia species* (esp. *M. restricta*), altered yeast load
Acne vulgaris	↓*Lactobacillus*, ↑*Proteobacteria*, ↓diversity	↑*Cutibacterium* acnes (formerly *Propionibacterium acnes*)

vitamin D levels positively correlating with the relative abundance of the *Lachnospira* and *Fusicatenibacter* genera of gut microbiome[76]. Vitamin D plays a key role in immune modulation, cell proliferation, differentiation, and maintaining intestinal homeostasis. Recent studies link vitamin D deficiency to increased IBD activity, highlighting its potential role in disease pathogenesis. Vitamin D receptor (VDR) signaling helps regulate gut immunity and preserve barrier integrity. VDR is abundantly expressed in healthy intestinal epithelial cells, especially in the crypts[77].

ROLE OF FAECAL MICROBIOTA TRANSPLANT IN GSA

Fecal microbiota transplantation (FMT) aims to restore gut dysbiosis and potentially improve outcomes in a variety of inflammatory conditions, including asthma and hidradenitis suppurativa[78]. However, the current evidence base for FMT in skin diseases remains limited to animal studies and human case reports[79]. Likewise, although faecal virome transplantation also has great potential in modulating the gut microbiome to suppress pro-inflammatory cascades, human trials on dermatological disorders are required. As research evolves, better-defined protocols and larger clinical studies will help clarify FMT's therapeutic potential for dermatological disorders[80].

FMT works by delivering a healthy donor's gut microbiome through faeces to a patient. Although FMT is established for certain infectious conditions, its role in dermatological disease remains exploratory, supported primarily by case reports and small pilot studies. The understanding of gut microbiota dysbiosis in chronic inflammatory skin diseases has expanded.

AD

Kim *et al*[81] first focused on employing FMT in AD therapy. FMT was tested for the first time at Tel Aviv Medical Center on adults with moderate to severe AD. In a total of nine participants, seven and six patients obtained a 50% and 75% reduction at week 18 (eight weeks following the last FMT), respectively. However, two of the patients had a fast relapse following treatment. The gut barrier is disrupted in AD patients, making them more sensitive to the dangers of FMT. Alternative gut microbiota -targeted therapeutics that include appropriate microbes or microbial metabolites may thus be a sensible strategy to treating AD in the future.

Psoriasis

The severity of psoriasis is linked to the decrease in gut microbiota diversity. The first clinical case report on FMT application detailed a 36-year-old Chinese man who had severe plaque psoriasis for a decade and IBD for fifteen years. At the end of five-week treatment with FMT, serum tumour necrosis factor-α levels, PASI, histology, and intestinal symptoms improved from baseline. The first proof-of-concept study, a parallel-group, double-blind, placebo-controlled, single-center superiority trial, including 31 participants, was conducted in 2015. The study found that FMT is safe but not more effective than sham for active peripheral psoriatic arthritis[82].

Alopecia areata

A pilot investigation observed alterations in the gut in pediatric patients with alopecia areata (AA) and discovered that *Ruminococcus bicirculans* abundance was lower in children (4-17 years) with AA compared to siblings without AA. There is no preventive treatment for AA. As a result, targeting the gut microbiota with FMT has emerged as a promising treatment for AA. Hair regrowth occurred after FMT in two individuals who had recurrent C. difficile infection with alopecia universalis. In an 86-year-old patient with patchy AA and non-infectious diarrhoea, hair regrew in the damaged places, and gray hairs gradually became black after FMT[83].

Systemic lupus erythematous

Research on FMT for systemic lupus erythematous in mice suggests that restoring gut microbiota with acidic water may help regulate disease progression. A 12-week pilot study in 20 systemic lupus erythematous patients showed that weekly oral FMT reduced SLEDAI-2K scores and anti-ds deoxy ribonucleic acid antibodies without major side effects[84].

FMT has also been studied in autoimmune conditions like Sjogren's and Behcet's disease, demonstrating potential in reducing chronic skin inflammation by restoring microbiome balance. However, challenges remain, including infection risks, donor-recipient compatibility, and regulatory hurdles, limiting its widespread adoption as a standardized treatment[84].

EMERGING INSIGHTS ON GSA

Earlier, microbial studies relied on culture-based methods, effective for common bacteria like *Staphylococcus aureus* but inadequate for fastidious ones like *Treponema pallidum*, limiting insights into the gut–skin microbiome. The introduction of 16S rRNA sequencing broadened analysis but couldn't fully capture viruses or provide precise bacterial quantification [85].

Shotgun metagenomics later enabled whole-genome sequencing, while advanced multi-omics approaches like meta-transcriptomics, meta-proteomics, and metabolomics offered deeper insights into microbial functions, enhancing our understanding of health and disease[86].

Research into biomarkers that bridge gut health and skin disease severity or treatment outcomes is still in its infancy. However, the premise that the gut microbiome can influence disease course is supported by studies demonstrating that faecal microbial composition can predict responses to PD-1-based immunotherapy for epithelial tumuors[87]. For instance, melanoma patients with different faecal microbiome profiles (particularly within the *ruminococcaceae* family) exhibit varying responses to anti-PD-1 therapies, and higher abundances of *B. longum*, *Collinsella aerofaciens,* and *Enterococcus faecium* were observed in responders to anti-PD-L1 therapy[88].

Separately, intestinal fatty acid binding protein and claudin-3, both biomarkers for gut permeability, are reportedly elevated in psoriasis and correlate with disease severity[89].

Studies examining the role of the gut microbiome in therapy response are scarce in dermatology, with most investigations focusing on IBD or rheumatologic autoimmune conditions. In rheumatoid arthritis, emerging data indicate that pre-treatment abundance of gut bacterial taxa and their genes are associated with future clinical response of methotrexate [90]. These human data add to the observations that germ-free or antibiotic-treated mice display reduced intestinal absorption and metabolism of methotrexate, underscoring the microbiome's vital role in drug biotransformation[91].

KNOWLEDGE GAPS IN THE GSA

Despite notable advances, it remains unclear whether microbiome imbalances directly drive inflammation in skin conditions such as psoriasis and AD or if they merely reflect a host environment already primed for inflammation[92]. While the gut microbiome has been extensively characterized, the specific mechanisms by which its metabolites and signalling molecules influence skin pathophysiology are still emerging[93].

The precise causal links between microbiome dysbiosis and disease progression are more clearly defined in some conditions, such as AD, than in others such as hidradenitis suppurativa and psoriasis. It also remains uncertain whether microbiome-associated findings represent true causative factors or are confounded by external environmental exposures.

On a general note, comparison of findings across different investigations is complicated by heterogeneity in sampling approaches (swabs *vs* biopsies), primer selection, and variations in sequencing protocols. Small sample sizes and insufficient sequencing depth also weaken the reliability of strain-level inferences, while diet and other lifestyle factors remain challenging to control[94,95]. Consequently, development of individualized microbiome modulation techniques is of major importance[96].

Finally, single cell engineered live biotherapeutic products are novel engineered live microorganisms to perform specific functions and represent a promising new avenue. Although tested primarily *in vitro* or *in vivo* so far, they hold great potential for refining GSA interventions and improving clinical outcomes[97].

INTEGRATED GASTROENTEROLOGY-DERMATOLOGY APPROACHES TO PATIENT MANAGEMENT

Inflammatory intestinal and skin diseases often share overlapping pathophysiology, present with highly variable clinical features, and carry increased risks of comorbid inflammatory conditions. These factors can result in diagnostic delays and challenges in distinguishing new disease processes from treatment side effects. In addition, chronic immune-mediated diseases have influential effects on co-occurring inflammatory diseases[98]. Dermatological manifestations may serve as early indicators of underlying GI pathology—such as aphthous ulcers in CD, or DH in celiac disease—and should prompt targeted investigations including endoscopy, serologic testing, or small bowel biopsy when appropriate. Establishing a formal, multidisciplinary collaboration between gastroenterologists and dermatologists can streamline care by enhancing awareness of associated conditions, facilitating prompt differential diagnoses, optimizing screening protocols, and optimizing therapeutic interventions[99]. Furthermore, shared patient registries and bio-banks should also be prioritized to advance research and inform joint management strategies. Ultimately, a unified therapeutic approach may reduce costs, minimize adverse events, and bolster patient adherence[100].

CONCLUSION

To fully harness the potential of GSA research and its clinical applications, a more coordinated, data-driven approach is essential. In particular, the following strategies warrant priority: (1) Standardized protocols: Harmonizing sample collection, sequencing, and analytic workflows will enable more direct comparisons and reproducible findings across studies; (2) Mechanistic investigations: Employing complementary *ex vivo*, *in vitro*, and clinical models will help elucidate precisely how bacterial strains modulate skin barrier function and inflammatory cascades. The role of viral, fungal, and archaea components are currently only emerging, and much research is needed to comprehensively understand the GSA; (3) Large-scale, longitudinal studies: Tracking well-characterized patient cohorts over time can capture dynamic microbial shifts and correlate these with clinical outcomes, disease relapses, and treatment responses; (4) *In situ* monitoring and microbiota recovery: Real-time assessment of skin microbes (*e.g.*, *via* non-invasive sampling) could facilitate personalized regimens aimed at isolating pathogenic strains, deciphering disease mechanisms, and guiding targeted probiotic or prebiotic interventions; and (5) Precision medicine and integrated omics: Incorporating genomic, micro-biomic, metabolomic, and pharmaco-micro-biomic profiles into machine-learning models will accelerate the development of individualized treatment strategies. This needs to also consider environmental factors, including dietary and lifestyle factors, along with host genetics. Notably, understanding how different therapeutic regimens interact with and reshape the microbiome—as both modulator and target—will be crucial for optimizing outcomes. Finally, there is a need for long-term follow-up studies to confirm durability and safety of beforementioned interventions. Collectively, these efforts have the potential to deepen our understanding of gut–skin crosstalk and transform patient care by moving beyond association studies toward mechanistic insights and precision-guided therapies.

ACKNOWLEDGEMENTS

Thanks to Anushka Saraswat for her assistance in creating the figures and tables.

FOOTNOTES

Author contributions: Singla N, Singla K, Attauabi M contributed to conceptualization, writing original draft, and revision; Aggarwal D contributed to methodology, assisted in the conceptualization, and contributed to the images and references; all authors read and approved the final manuscript.

Conflict-of-interest statement: All the authors report no relevant conflicts of interest for this article.

Country of origin: Denmark

ORCID number: Neeraj Singla 0000-0003-1007-7477; Mohamed Attauabi 0000-0003-0323-7030.

S-Editor: Liu H
L-Editor: A
P-Editor: Zhang XD

REFERENCES

1 **Williams RE**. Benefit and mischief from commensal bacteria. *J Clin Pathol* 1973; **26**: 811-818 [*RCA*] [PMID: 4271885 DOI: 10.1136/jcp.26.11.811] [FullText]
2 **Savage DC**. Microbial ecology of the gastrointestinal tract. *Annu Rev Microbiol* 1977; **31**: 107-133 [*RCA*] [PMID: 334036 DOI: 10.1146/annurev.mi.31.100177.000543] [FullText]
3 **De Pessemier B**, Grine L, Debaere M, Maes A, Paetzold B, Callewaert C. Gut-Skin Axis: Current Knowledge of the Interrelationship between Microbial Dysbiosis and Skin Conditions. *Microorganisms* 2021; **9**: 353 [*RCA*] [PMID: 33670115 DOI: 10.3390/microorganisms9020353] [FullText] [Full Text(PDF)]
4 **Grice EA**, Segre JA. The skin microbiome. *Nat Rev Microbiol* 2011; **9**: 244-253 [*RCA*] [PMID: 21407241 DOI: 10.1038/nrmicro2537] [Full Text] [Full Text(PDF)]
5 **Shah KR**, Boland CR, Patel M, Thrash B, Menter A. Cutaneous manifestations of gastrointestinal disease: part I. *J Am Acad Dermatol* 2013; **68**: 189.e1-21; quiz 210 [*RCA*] [PMID: 23317980 DOI: 10.1016/j.jaad.2012.10.037] [FullText]
6 **Rahvar M**, Kerstetter J. Cutaneous manifestation of gastrointestinal disease. *J Gastrointest Oncol* 2016; **7**: S44-S54 [*RCA*] [PMID: 27034812 DOI: 10.3978/j.issn.2078-6891.2015.059] [FullText]
7 **Gallo RL**. Human Skin Is the Largest Epithelial Surface for Interaction with Microbes. *J Invest Dermatol* 2017; **137**: 1213-1214 [*RCA*]

[PMID: 28395897 DOI: 10.1016/j.jid.2016.11.045] [FullText]
8 **O'Neill CA**, Monteleone G, McLaughlin JT, Paus R. The gut-skin axis in health and disease: A paradigm with therapeutic implications. *Bioessays* 2016; **38**: 1167-1176 [*RCA*] [PMID: 27554239 DOI: 10.1002/bies.201600008] [FullText]
9 **Levkovich T**, Poutahidis T, Smillie C, Varian BJ, Ibrahim YM, Lakritz JR, Alm EJ, Erdman SE. Probiotic bacteria induce a 'glow of health'. *PLoS One* 2013; **8**: e53867 [*RCA*] [PMID: 23342023 DOI: 10.1371/journal.pone.0053867] [FullText] [Full Text(PDF)]
10 **Vojvodic A**, Peric-Hajzler Z, Matovic D, Vojvodic P, Vlaskovic-Jovicevic T, Sijan G, Dimitrijevic S, Stepic N, Wollina U, Badr BAE, Badawi A, Goldust M, Tirant M, Nguyen VT, Fioranelli M, Lotti T. Gut Microbiota and the Alteration of Immune Balance in Skin Diseases: From Nutraceuticals to Fecal Transplantation. *Open Access Maced J Med Sci* 2019; **7**: 3034-3038 [*RCA*] [PMID: 31850117 DOI: 10.3889/oamjms.2019.827] [FullText] [Full Text(PDF)]
11 **NIH HMP Working Group**, Peterson J, Garges S, Giovanni M, McInnes P, Wang L, Schloss JA, Bonazzi V, McEwen JE, Wetterstrand KA, Deal C, Baker CC, Di Francesco V, Howcroft TK, Karp RW, Lunsford RD, Wellington CR, Belachew T, Wright M, Giblin C, David H, Mills M, Salomon R, Mullins C, Akolkar B, Begg L, Davis C, Grandison L, Humble M, Khalsa J, Little AR, Peavy H, Pontzer C, Portnoy M, Sayre MH, Starke-Reed P, Zakhari S, Read J, Watson B, Guyer M. The NIH Human Microbiome Project. *Genome Res* 2009; **19**: 2317-2323 [*RCA*] [PMID: 19819907 DOI: 10.1101/gr.096651.109] [FullText]
12 **Arck P**, Handjiski B, Hagen E, Pincus M, Bruenahl C, Bienenstock J, Paus R. Is there a 'gut-brain-skin axis'? *Exp Dermatol* 2010; **19**: 401-405 [*RCA*] [PMID: 20113345 DOI: 10.1111/j.1600-0625.2009.01060.x] [FullText]
13 **Ellis SR**, Nguyen M, Vaughn AR, Notay M, Burney WA, Sandhu S, Sivamani RK. The Skin and Gut Microbiome and Its Role in Common Dermatologic Conditions. *Microorganisms* 2019; **7**: 550 [*RCA*] [PMID: 31717915 DOI: 10.3390/microorganisms7110550] [FullText] [Full Text(PDF)]
14 **Salem I**, Ramser A, Isham N, Ghannoum MA. The Gut Microbiome as a Major Regulator of the Gut-Skin Axis. *Front Microbiol* 2018; **9**: 1459 [*RCA*] [PMID: 30042740 DOI: 10.3389/fmicb.2018.01459] [FullText] [Full Text(PDF)]
15 **Maguire M**, Maguire G. The role of microbiota, and probiotics and prebiotics in skin health. *Arch Dermatol Res* 2017; **309**: 411-421 [*RCA*] [PMID: 28631091 DOI: 10.1007/s00403-017-1750-3] [FullText]
16 **Miyazaki K**, Masuoka N, Kano M, Iizuka R. Bifidobacterium fermented milk and galacto-oligosaccharides lead to improved skin health by decreasing phenols production by gut microbiota. *Benef Microbes* 2014; **5**: 121-128 [*RCA*] [PMID: 23685373 DOI: 10.3920/BM2012.0066] [FullText]
17 **Lyte M**. Microbial endocrinology and the microbiota-gut-brain axis. *Adv Exp Med Biol* 2014; **817**: 3-24 [*RCA*] [PMID: 24997027 DOI: 10.1007/978-1-4939-0897-4_1] [FullText]
18 **Rea K**, Dinan TG, Cryan JF. The microbiome: A key regulator of stress and neuroinflammation. *Neurobiol Stress* 2016; **4**: 23-33 [*RCA*] [PMID: 27981187 DOI: 10.1016/j.ynstr.2016.03.001] [FullText] [Full Text(PDF)]
19 **Islam SA**, Luster AD. T cell homing to epithelial barriers in allergic disease. *Nat Med* 2012; **18**: 705-715 [*RCA*] [PMID: 22561834 DOI: 10.1038/nm.2760] [FullText]
20 **Kulig M**, Bergmann R, Klettke U, Wahn V, Tacke U, Wahn U. Natural course of sensitization to food and inhalant allergens during the first 6 years of life. *J Allergy Clin Immunol* 1999; **103**: 1173-1179 [*RCA*] [PMID: 10359902 DOI: 10.1016/s0091-6749(99)70195-8] [FullText]
21 **Du Toit G**, Roberts G, Sayre PH, Bahnson HT, Radulovic S, Santos AF, Brough HA, Phippard D, Basting M, Feeney M, Turcanu V, Sever ML, Gomez Lorenzo M, Plaut M, Lack G; LEAP Study Team. Randomized trial of peanut consumption in infants at risk for peanut allergy. *N Engl J Med* 2015; **372**: 803-813 [*RCA*] [PMID: 25705822 DOI: 10.1056/NEJMoa1414850] [FullText]
22 **Bertolani M**, Rodighiero E, Saleri R, Pedrazzi G, Bertoli S, Leone A, Feliciani C, Lotti T, Satolli F. The influence of Mediterranean diet in acne pathogenesis and the correlation with insulin-like growth factor-1 serum levels: Implications and results. *Dermatol Reports* 2022; **14**: 9143 [*RCA*] [PMID: 35371424 DOI: 10.4081/dr.2022.9143] [FullText] [Full Text(PDF)]
23 **Wahlström A**, Sayin SI, Marschall HU, Bäckhed F. Intestinal Crosstalk between Bile Acids and Microbiota and Its Impact on Host Metabolism. *Cell Metab* 2016; **24**: 41-50 [*RCA*] [PMID: 27320064 DOI: 10.1016/j.cmet.2016.05.005] [FullText]
24 **Widhiati S**, Purnomosari D, Wibawa T, Soebono H. The role of gut microbiome in inflammatory skin disorders: A systematic review. *Dermatol Reports* 2022; **14**: 9188 [*RCA*] [PMID: 35371420 DOI: 10.4081/dr.2022.9188] [FullText] [Full Text(PDF)]
25 **Passmore IJ**, Letertre MPM, Preston MD, Bianconi I, Harrison MA, Nasher F, Kaur H, Hong HA, Baines SD, Cutting SM, Swann JR, Wren BW, Dawson LF. Para-cresol production by Clostridium difficile affects microbial diversity and membrane integrity of Gram-negative bacteria. *PLoS Pathog* 2018; **14**: e1007191 [*RCA*] [PMID: 30208103 DOI: 10.1371/journal.ppat.1007191] [FullText] [Full Text(PDF)]
26 **Cho M**, Woo YR, Cho SH, Lee JD, Kim HS. Metformin: A Potential Treatment for Acne, Hidradenitis Suppurativa and Rosacea. *Acta Derm Venereol* 2023; **103**: adv18392 [*RCA*] [PMID: 38078688 DOI: 10.2340/actadv.v103.18392] [FullText]
27 **Carlessi AS**, Borba LA, Zugno AI, Quevedo J, Réus GZ. Gut microbiota-brain axis in depression: The role of neuroinflammation. *Eur J Neurosci* 2021; **53**: 222-235 [*RCA*] [PMID: 31785168 DOI: 10.1111/ejn.14631] [FullText]
28 **Dinan TG**, Cryan JF. The Microbiome-Gut-Brain Axis in Health and Disease. *Gastroenterol Clin North Am* 2017; **46**: 77-89 [*RCA*] [PMID: 28164854 DOI: 10.1016/j.gtc.2016.09.007] [FullText]
29 **Slominski A**, Wortsman J. Neuroendocrinology of the skin. *Endocr Rev* 2000; **21**: 457-487 [*RCA*] [PMID: 11041445 DOI: 10.1210/edrv.21.5.0410] [FullText]
30 **Lankarani KB**, Sivandzadeh GR, Hassanpour S. Oral manifestation in inflammatory bowel disease: a review. *World J Gastroenterol* 2013; **19**: 8571-8579 [*RCA*] [PMID: 24379574 DOI: 10.3748/wjg.v19.i46.8571] [FullText] [Full Text(PDF)]
31 **Turkcapar N**, Toruner M, Soykan I, Aydintug OT, Cetinkaya H, Duzgun N, Ozden A, Duman M. The prevalence of extraintestinal manifestations and HLA association in patients with inflammatory bowel disease. *Rheumatol Int* 2006; **26**: 663-668 [*RCA*] [PMID: 16136311 DOI: 10.1007/s00296-005-0044-9] [FullText]
32 **Weinstein M**, Turner D, Avitzur Y. Erythema nodosum as a presentation of inflammatory bowel disease. *CMAJ* 2005; **173**: 145-146 [*RCA*] [PMID: 16027427 DOI: 10.1503/cmaj.050201] [FullText]
33 **Marzano AV**, Borghi A, Stadnicki A, Crosti C, Cugno M. Cutaneous manifestations in patients with inflammatory bowel diseases: pathophysiology, clinical features, and therapy. *Inflamm Bowel Dis* 2014; **20**: 213-227 [*RCA*] [PMID: 24105394 DOI: 10.1097/01.MIB.0000436959.62286.f9] [FullText]
34 **Pérez-Garza DM**, Chavez-Alvarez S, Ocampo-Candiani J, Gomez-Flores M. Erythema Nodosum: A Practical Approach and Diagnostic Algorithm. *Am J Clin Dermatol* 2021; **22**: 367-378 [*RCA*] [PMID: 33683567 DOI: 10.1007/s40257-021-00592-w] [FullText] [Full Text(PDF)]
35 **Antonelli E**, Bassotti G, Tramontana M, Hansel K, Stingeni L, Ardizzone S, Genovese G, Marzano AV, Maconi G. Dermatological

Manifestations in Inflammatory Bowel Diseases. *J Clin Med* 2021; **10**: 364 [*RCA*] [PMID: 33477990 DOI: 10.3390/jcm10020364] [FullText] [Full Text(PDF)]

36 **George C**, Deroide F, Rustin M. Pyoderma gangrenosum - a guide to diagnosis and management. *Clin Med (Lond)* 2019; **19**: 224-228 [*RCA*] [PMID: 31092515 DOI: 10.7861/clinmedicine.19-3-224] [FullText]

37 **Plumptre I**, Knabel D, Tomecki K. Pyoderma Gangrenosum: A Review for the Gastroenterologist. *Inflamm Bowel Dis* 2018; **24**: 2510-2517 [*RCA*] [PMID: 29788368 DOI: 10.1093/ibd/izy174] [FullText]

38 **Saghafi S**, Falaki F, Bashardoost N. Pyostomatitis vegetans: report of a rare case. *Pol J Pathol* 2011; **62**: 125-128 [*RCA*] [PMID: 21866474] [FullText]

39 **Atarbashi-Moghadam S**, Lotfi A, Atarbashi-Moghadam F. Pyostomatitis Vegetans: A Clue for Diagnosis of Silent Crohn's Disease. *J Clin Diagn Res* 2016; **10**: ZD12-ZD13 [*RCA*] [PMID: 28209014 DOI: 10.7860/JCDR/2016/22573.9032] [FullText]

40 **Sbeit W**, Kadah A, Mahamid M, Karayanni H, Mari A, Tali S, Srouji S, Khoury T. Oral manifestations of inflammatory bowel disease: the neglected piece of the puzzle. *Eur J Gastroenterol Hepatol* 2020; **32**: 1422-1431 [*RCA*] [PMID: 32925508 DOI: 10.1097/MEG.0000000000001918] [FullText]

41 **Zunt SL**. Recurrent aphthous stomatitis. *Dermatol Clin* 2003; **21**: 33-39 [*RCA*] [PMID: 12622266 DOI: 10.1016/s0733-8635(02)00075-x] [FullText]

42 **Gasmi Benahmed A**, Noor S, Menzel A, Gasmi A. Oral Aphthous: Pathophysiology, Clinical Aspects and Medical Treatment. *Arch Razi Inst* 2021; **76**: 1155-1163 [*RCA*] [PMID: 35355774 DOI: 10.22092/ari.2021.356055.1767] [FullText]

43 **Alinaghi F**, Tekin HG, Burisch J, Wu JJ, Thyssen JP, Egeberg A. Global Prevalence and Bidirectional Association Between Psoriasis and Inflammatory Bowel Disease-A Systematic Review and Meta-analysis. *J Crohns Colitis* 2020; **14**: 351-360 [*RCA*] [PMID: 31504363 DOI: 10.1093/ecco-jcc/jjz152] [FullText]

44 **Hedin CRH**, Sonkoly E, Eberhardson M, Ståhle M. Inflammatory bowel disease and psoriasis: modernizing the multidisciplinary approach. *J Intern Med* 2021; **290**: 257-278 [*RCA*] [PMID: 33942408 DOI: 10.1111/joim.13282] [FullText]

45 **Antiga E**, Caproni M. The diagnosis and treatment of dermatitis herpetiformis. *Clin Cosmet Investig Dermatol* 2015; **8**: 257-265 [*RCA*] [PMID: 25999753 DOI: 10.2147/CCID.S69127] [FullText] [Full Text(PDF)]

46 **Persechino F**, Galli G, Persechino S, Valitutti F, Zenzeri L, Mauro A, Corleto VD, Parisi P, Ziparo C, Evangelisti M, Quatrale G, Di Nardo G. Skin Manifestations and Coeliac Disease in Paediatric Population. *Nutrients* 2021; **13**: 3611 [*RCA*] [PMID: 34684612 DOI: 10.3390/nu13103611] [FullText] [Full Text(PDF)]

47 **Caproni M**, Bonciolini V, D'Errico A, Antiga E, Fabbri P. Celiac disease and dermatologic manifestations: many skin clue to unfold gluten-sensitive enteropathy. *Gastroenterol Res Pract* 2012; **2012**: 952753 [*RCA*] [PMID: 22693492 DOI: 10.1155/2012/952753] [FullText] [Full Text(PDF)]

48 **Jo YW**, Min JH, Kwon JW, Her Y. Rosacea and Its Potential Role in the Development of Irritable Bowel Syndrome: Insights From the Korean National Health Insurance Service-National Sample Cohort. *J Korean Med Sci* 2025; **40**: e97 [*RCA*] [PMID: 40461142 DOI: 10.3346/jkms.2025.40.e97] [FullText] [Full Text(PDF)]

49 **Chiesa Fuxench ZC**, Wan J, Wang S, Syed MN, Shin DB, Abuabara K, Gelfand JM. Risk of Inflammatory Bowel Disease in Patients With Atopic Dermatitis. *JAMA Dermatol* 2023; **159**: 1085-1092 [*RCA*] [PMID: 37647058 DOI: 10.1001/jamadermatol.2023.2875] [FullText]

50 **Blicharz L**, Michalak M, Szymanek-Majchrzak K, Młynarczyk G, Skowroński K, Rudnicka L, Samochocki Z. The Propensity to Form Biofilm in vitro by Staphylococcus aureus Strains Isolated from the Anterior Nares of Patients with Atopic Dermatitis: Clinical Associations. *Dermatology* 2021; **237**: 528-534 [*RCA*] [PMID: 33113538 DOI: 10.1159/000511182] [FullText]

51 **Song H**, Yoo Y, Hwang J, Na YC, Kim HS. Faecalibacterium prausnitzii subspecies-level dysbiosis in the human gut microbiome underlying atopic dermatitis. *J Allergy Clin Immunol* 2016; **137**: 852-860 [*RCA*] [PMID: 26431583 DOI: 10.1016/j.jaci.2015.08.021] [FullText]

52 **Abrahamsson TR**, Jakobsson HE, Andersson AF, Björkstén B, Engstrand L, Jenmalm MC. Low gut microbiota diversity in early infancy precedes asthma at school age. *Clin Exp Allergy* 2014; **44**: 842-850 [*RCA*] [PMID: 24330256 DOI: 10.1111/cea.12253] [FullText]

53 **Bjerre RD**, Holm JB, Palleja A, Sølberg J, Skov L, Johansen JD. Skin dysbiosis in the microbiome in atopic dermatitis is site-specific and involves bacteria, fungus and virus. *BMC Microbiol* 2021; **21**: 256 [*RCA*] [PMID: 34551705 DOI: 10.1186/s12866-021-02302-2] [FullText] [Full Text(PDF)]

54 **Devereux G**, Seaton A. Diet as a risk factor for atopy and asthma. *J Allergy Clin Immunol* 2005; **115**: 1109-17; quiz 1118 [*RCA*] [PMID: 15940119 DOI: 10.1016/j.jaci.2004.12.1139] [FullText]

55 **Fry L**, Baker BS. Triggering psoriasis: the role of infections and medications. *Clin Dermatol* 2007; **25**: 606-615 [*RCA*] [PMID: 18021899 DOI: 10.1016/j.clindermatol.2007.08.015] [FullText]

56 **Alekseyenko AV**, Perez-Perez GI, De Souza A, Strober B, Gao Z, Bihan M, Li K, Methé BA, Blaser MJ. Community differentiation of the cutaneous microbiota in psoriasis. *Microbiome* 2013; **1**: 31 [*RCA*] [PMID: 24451201 DOI: 10.1186/2049-2618-1-31] [FullText] [Full Text (PDF)]

57 **Chen YJ**, Ho HJ, Tseng CH, Lai ZL, Shieh JJ, Wu CY. Intestinal microbiota profiling and predicted metabolic dysregulation in psoriasis patients. *Exp Dermatol* 2018; **27**: 1336-1343 [*RCA*] [PMID: 30238519 DOI: 10.1111/exd.13786] [FullText]

58 **Lao J**, Xie Z, Qin Q, Qin R, Li S, Yuan Y. Serum LL-37 and inflammatory cytokines levels in psoriasis. *Immun Inflamm Dis* 2023; **11**: e802 [*RCA*] [PMID: 36988247 DOI: 10.1002/iid3.802] [FullText]

59 **Zaidi AK**, Spaunhurst K, Sprockett D, Thomason Y, Mann MW, Fu P, Ammons C, Gerstenblith M, Tuttle MS, Popkin DL. Characterization of the facial microbiome in twins discordant for rosacea. *Exp Dermatol* 2018; **27**: 295-298 [*RCA*] [PMID: 29283459 DOI: 10.1111/exd.13491] [FullText]

60 **Thompson KG**, Rainer BM, Kang S, Chien AL. The skin microbiota as a link between rosacea and its systemic comorbidities. *Int J Dermatol* 2020; **59**: 513-514 [*RCA*] [PMID: 32043275 DOI: 10.1111/ijd.14802] [FullText]

61 **Moreno-Arrones OM**, Ortega-Quijano D, Perez-Brocal V, Fernandez-Nieto D, Jimenez N, de Las Heras E, Moya A, Perez-Garcia B. Dysbiotic gut microbiota in patients with inflammatory rosacea: another clue towards the existence of a brain-gut-skin axis. *Br J Dermatol* 2021; **185**: 655-657 [*RCA*] [PMID: 33913159 DOI: 10.1111/bjd.20411] [FullText]

62 **Jfri A**, Nassim D, O'Brien E, Gulliver W, Nikolakis G, Zouboulis CC. Prevalence of Hidradenitis Suppurativa: A Systematic Review and Meta-regression Analysis. *JAMA Dermatol* 2021; **157**: 924-931 [*RCA*] [PMID: 34037678 DOI: 10.1001/jamadermatol.2021.1677] [FullText]

63 **McCarthy S**, Barrett M, Kirthi S, Pellanda P, Vlckova K, Tobin AM, Murphy M, Shanahan F, O'Toole PW. Altered Skin and Gut Microbiome in Hidradenitis Suppurativa. *J Invest Dermatol* 2022; **142**: 459-468.e15 [*RCA*] [PMID: 34364884 DOI: 10.1016/j.jid.2021.05.036] [FullText]

64 **Chen YH**, Wu CS, Chao YH, Lin CC, Tsai HY, Li YR, Chen YZ, Tsai WH, Chen YK. Lactobacillus pentosus GMNL-77 inhibits skin lesions in imiquimod-induced psoriasis-like mice. *J Food Drug Anal* 2017; **25**: 559-566 [*RCA*] [PMID: 28911642 DOI: 10.1016/j.jfda.2016.06.003] [FullText] [Full Text(PDF)]

65 **Konieczna P**, Akdis CA, Quigley EM, Shanahan F, O'Mahony L. Portrait of an immunoregulatory Bifidobacterium. *Gut Microbes* 2012; **3**: 261-266 [*RCA*] [PMID: 22572827 DOI: 10.4161/gmic.20358] [FullText]

66 **Groeger D**, O'Mahony L, Murphy EF, Bourke JF, Dinan TG, Kiely B, Shanahan F, Quigley EM. Bifidobacterium infantis 35624 modulates host inflammatory processes beyond the gut. *Gut Microbes* 2013; **4**: 325-339 [*RCA*] [PMID: 23842110 DOI: 10.4161/gmic.25487] [FullText] [Full Text(PDF)]

67 **Moludi J**, Khedmatgozar H, Saiedi S, Razmi H, Alizadeh M, Ebrahimi B. Probiotic supplementation improves clinical outcomes and quality of life indicators in patients with plaque psoriasis: A randomized double-blind clinical trial. *Clin Nutr ESPEN* 2021; **46**: 33-39 [*RCA*] [PMID: 34857215 DOI: 10.1016/j.clnesp.2021.09.004] [FullText]

68 **Lin C**, Zeng T, Deng Y, Yang W, Xiong J. [Treatment of psoriasis vulgaris using Bacteroides fragilis BF839: a single-arm, open preliminary clinical study]. *Sheng Wu Gong Cheng Xue Bao* 2021; **37**: 3828-3835 [*RCA*] [PMID: 34841788 DOI: 10.13345/j.cjb.210198] [FullText]

69 **Parodi A**, Paolino S, Greco A, Drago F, Mansi C, Rebora A, Parodi A, Savarino V. Small intestinal bacterial overgrowth in rosacea: clinical effectiveness of its eradication. *Clin Gastroenterol Hepatol* 2008; **6**: 759-764 [*RCA*] [PMID: 18456568 DOI: 10.1016/j.cgh.2008.02.054] [Full Text]

70 **Ma YY**, Li L, Yu CH, Shen Z, Chen LH, Li YM. Effects of probiotics on nonalcoholic fatty liver disease: a meta-analysis. *World J Gastroenterol* 2013; **19**: 6911-6918 [*RCA*] [PMID: 24187469 DOI: 10.3748/wjg.v19.i40.6911] [FullText] [Full Text(PDF)]

71 **Navarro-López V**, Ramírez-Boscá A, Ramón-Vidal D, Ruzafa-Costas B, Genovés-Martínez S, Chenoll-Cuadros E, Carrión-Gutiérrez M, Horga de la Parte J, Prieto-Merino D, Codoñer-Cortés FM. Effect of Oral Administration of a Mixture of Probiotic Strains on SCORAD Index and Use of Topical Steroids in Young Patients With Moderate Atopic Dermatitis: A Randomized Clinical Trial. *JAMA Dermatol* 2018; **154**: 37-43 [*RCA*] [PMID: 29117309 DOI: 10.1001/jamadermatol.2017.3647] [FullText]

72 **Cuello-Garcia C**, Fiocchi A, Pawankar R, Yepes-Nuñez JJ, Morgano GP, Zhang Y, Agarwal A, Gandhi S, Terracciano L, Schünemann HJ, Brozek JL. Prebiotics for the prevention of allergies: A systematic review and meta-analysis of randomized controlled trials. *Clin Exp Allergy* 2017; **47**: 1468-1477 [*RCA*] [PMID: 29035013 DOI: 10.1111/cea.13042] [FullText]

73 **Sinha S**, Lin G, Ferenczi K. The skin microbiome and the gut-skin axis. *Clin Dermatol* 2021; **39**: 829-839 [*RCA*] [PMID: 34785010 DOI: 10.1016/j.clindermatol.2021.08.021] [FullText]

74 **Cuello-Garcia CA**, Fiocchi A, Pawankar R, Yepes-Nuñez JJ, Morgano GP, Zhang Y, Ahn K, Al-Hammadi S, Agarwal A, Gandhi S, Beyer K, Burks W, Canonica GW, Ebisawa M, Kamenwa R, Lee BW, Li H, Prescott S, Riva JJ, Rosenwasser L, Sampson H, Spigler M, Terracciano L, Vereda A, Waserman S, Schünemann HJ, Brożek JL. World Allergy Organization-McMaster University Guidelines for Allergic Disease Prevention (GLAD-P): Prebiotics. *World Allergy Organ J* 2016; **9**: 10 [*RCA*] [PMID: 26962387 DOI: 10.1186/s40413-016-0102-7] [FullText] [Full Text(PDF)]

75 **Pelucchi C**, Chatenoud L, Turati F, Galeone C, Moja L, Bach JF, La Vecchia C. Probiotics supplementation during pregnancy or infancy for the prevention of atopic dermatitis: a meta-analysis. *Epidemiology* 2012; **23**: 402-414 [*RCA*] [PMID: 22441545 DOI: 10.1097/EDE.0b013e31824d5da2] [FullText]

76 **Bosman ES**, Albert AY, Lui H, Dutz JP, Vallance BA. Skin Exposure to Narrow Band Ultraviolet (UVB) Light Modulates the Human Intestinal Microbiome. *Front Microbiol* 2019; **10**: 2410 [*RCA*] [PMID: 31708890 DOI: 10.3389/fmicb.2019.02410] [FullText] [Full Text (PDF)]

77 **Aggeletopoulou I**, Marangos M, Assimakopoulos SF, Mouzaki A, Thomopoulos K, Triantos C. Vitamin D and Microbiome: Molecular Interaction in Inflammatory Bowel Disease Pathogenesis. *Am J Pathol* 2023; **193**: 656-668 [*RCA*] [PMID: 36868465 DOI: 10.1016/j.ajpath.2023.02.004] [FullText]

78 **Mashiah J**, Karady T, Fliss-Isakov N, Sprecher E, Slodownik D, Artzi O, Samuelov L, Ellenbogen E, Godneva A, Segal E, Maharshak N. Clinical efficacy of fecal microbial transplantation treatment in adults with moderate-to-severe atopic dermatitis. *Immun Inflamm Dis* 2022; **10**: e570 [*RCA*] [PMID: 34931478 DOI: 10.1002/iid3.570] [FullText] [Full Text(PDF)]

79 **Zou X**, Zou X, Gao L, Zhao H. Gut microbiota and psoriasis: pathogenesis, targeted therapy, and future directions. *Front Cell Infect Microbiol* 2024; **14**: 1430586 [*RCA*] [PMID: 39170985 DOI: 10.3389/fcimb.2024.1430586] [FullText]

80 **Jansen D**, Matthijnssens J. The Emerging Role of the Gut Virome in Health and Inflammatory Bowel Disease: Challenges, Covariates and a Viral Imbalance. *Viruses* 2023; **15**: 173 [*RCA*] [PMID: 36680214 DOI: 10.3390/v15010173] [FullText] [Full Text(PDF)]

81 **Kim JH**, Kim K, Kim W. Gut microbiota restoration through fecal microbiota transplantation: a new atopic dermatitis therapy. *Exp Mol Med* 2021; **53**: 907-916 [*RCA*] [PMID: 34017060 DOI: 10.1038/s12276-021-00627-6] [FullText] [Full Text(PDF)]

82 **Wu M**, Chen X, Lu Q, Yao X. Fecal microbiota transplantation for the treatment of chronic inflammatory skin diseases. *Heliyon* 2024; **10**: e37432 [*RCA*] [PMID: 39309854 DOI: 10.1016/j.heliyon.2024.e37432] [FullText] [Full Text(PDF)]

83 **Xie WR**, Yang XY, Xia HH, Wu LH, He XX. Hair regrowth following fecal microbiota transplantation in an elderly patient with alopecia areata: A case report and review of the literature. *World J Clin Cases* 2019; **7**: 3074-3081 [*RCA*] [PMID: 31624757 DOI: 10.12998/wjcc.v7.i19.3074] [FullText] [Full Text(PDF)]

84 **Huang C**, Yi P, Zhu M, Zhou W, Zhang B, Yi X, Long H, Zhang G, Wu H, Tsokos GC, Zhao M, Lu Q. Safety and efficacy of fecal microbiota transplantation for treatment of systemic lupus erythematosus: An EXPLORER trial. *J Autoimmun* 2022; **130**: 102844 [*RCA*] [PMID: 35690527 DOI: 10.1016/j.jaut.2022.102844] [FullText]

85 **Turnbaugh PJ**, Ley RE, Hamady M, Fraser-Liggett CM, Knight R, Gordon JI. The human microbiome project. *Nature* 2007; **449**: 804-810 [*RCA*] [PMID: 17943116 DOI: 10.1038/nature06244] [FullText] [Full Text(PDF)]

86 **Roux PF**, Oddos T, Stamatas G. Deciphering the Role of Skin Surface Microbiome in Skin Health: An Integrative Multiomics Approach Reveals Three Distinct Metabolite–Microbe Clusters. *J Invest Dermatol* 2022; **142**: 469-479.e5 [*RCA*] [PMID: 34343557 DOI: 10.1016/j.jid.2021.07.159] [FullText]

87 **Routy B**, Le Chatelier E, Derosa L, Duong CPM, Alou MT, Daillère R, Fluckiger A, Messaoudene M, Rauber C, Roberti MP, Fidelle M, Flament C, Poirier-Colame V, Opolon P, Klein C, Iribarren K, Mondragón L, Jacquelot N, Qu B, Ferrere G, Clémenson C, Mezquita L, Masip JR, Naltet C, Brosseau S, Kaderbhai C, Richard C, Rizvi H, Levenez F, Galleron N, Quinquis B, Pons N, Ryffel B, Minard-Colin V, Gonin P, Soria JC, Deutsch E, Loriot Y, Ghiringhelli F, Zalcman G, Goldwasser F, Escudier B, Hellmann MD, Eggermont A, Raoult D, Albiges L, Kroemer G, Zitvogel L. Gut microbiome influences efficacy of PD-1-based immunotherapy against epithelial tumors. *Science* 2018; **359**: 91-97

[*RCA*] [PMID: 29097494 DOI: 10.1126/science.aan3706] [FullText]

88 **Matson V**, Fessler J, Bao R, Chongsuwat T, Zha Y, Alegre ML, Luke JJ, Gajewski TF. The commensal microbiome is associated with anti-PD-1 efficacy in metastatic melanoma patients. *Science* 2018; **359**: 104-108 [*RCA*] [PMID: 29302014 DOI: 10.1126/science.aao3290] [Full Text]

89 **Sikora M**, Stec A, Chrabaszcz M, Waskiel-Burnat A, Zaremba M, Olszewska M, Rudnicka L. Intestinal Fatty Acid Binding Protein, a Biomarker of Intestinal Barrier, is Associated with Severity of Psoriasis. *J Clin Med* 2019; **8**: 1021 [*RCA*] [PMID: 31336842 DOI: 10.3390/jcm8071021] [FullText] [Full Text(PDF)]

90 **Artacho A**, Isaac S, Nayak R, Flor-Duro A, Alexander M, Koo I, Manasson J, Smith PB, Rosenthal P, Homsi Y, Gulko P, Pons J, Puchades-Carrasco L, Izmirly P, Patterson A, Abramson SB, Pineda-Lucena A, Turnbaugh PJ, Ubeda C, Scher JU. The Pretreatment Gut Microbiome Is Associated With Lack of Response to Methotrexate in New-Onset Rheumatoid Arthritis. *Arthritis Rheumatol* 2021; **73**: 931-942 [*RCA*] [PMID: 33314800 DOI: 10.1002/art.41622] [FullText]

91 **Valerino DM**, Johns DG, Zaharko DS, Oliverio VT. Studies of the metabolism of methotrexate by intestinal flora. I. Identification and study of biological properties of the metabolite 4-amino-4-deoxy-N 10 -methylpteroic acid. *Biochem Pharmacol* 1972; **21**: 821-831 [*RCA*] [PMID: 5014749 DOI: 10.1016/0006-2952(72)90125-6] [FullText]

92 **Chen Y**, Knight R, Gallo RL. Evolving approaches to profiling the microbiome in skin disease. *Front Immunol* 2023; **14**: 1151527 [*RCA*] [PMID: 37081873 DOI: 10.3389/fimmu.2023.1151527] [FullText]

93 **Zhu Q**, Wu K, Yang Q, Meng B, Niu Y, Zhao F. Advances in psoriasis and gut microorganisms with co-metabolites. *Front Microbiol* 2023; **14**: 1192543 [*RCA*] [PMID: 38033573 DOI: 10.3389/fmicb.2023.1192543] [FullText]

94 **Chen L**, Li J, Zhu W, Kuang Y, Liu T, Zhang W, Chen X, Peng C. Skin and Gut Microbiome in Psoriasis: Gaining Insight Into the Pathophysiology of It and Finding Novel Therapeutic Strategies. *Front Microbiol* 2020; **11**: 589726 [*RCA*] [PMID: 33384669 DOI: 10.3389/fmicb.2020.589726] [FullText] [Full Text(PDF)]

95 **Sikora M**, Stec A, Chrabaszcz M, Knot A, Waskiel-Burnat A, Rakowska A, Olszewska M, Rudnicka L. Gut Microbiome in Psoriasis: An Updated Review. *Pathogens* 2020; **9**: 463 [*RCA*] [PMID: 32545459 DOI: 10.3390/pathogens9060463] [FullText] [Full Text(PDF)]

96 **Chen M**, Wang R, Wang T. Gut microbiota and skin pathologies: Mechanism of the gut-skin axis in atopic dermatitis and psoriasis. *Int Immunopharmacol* 2024; **141**: 112658 [*RCA*] [PMID: 39137625 DOI: 10.1016/j.intimp.2024.112658] [FullText]

97 **Rutter JW**, Dekker L, Owen KA, Barnes CP. Microbiome engineering: engineered live biotherapeutic products for treating human disease. *Front Bioeng Biotechnol* 2022; **10**: 1000873 [*RCA*] [PMID: 36185459 DOI: 10.3389/fbioe.2022.1000873] [FullText] [Full Text(PDF)]

98 **Attauabi M**, Zhao M, Bendtsen F, Burisch J. Systematic Review with Meta-analysis: The Impact of Co-occurring Immune-mediated Inflammatory Diseases on the Disease Course of Inflammatory Bowel Diseases. *Inflamm Bowel Dis* 2021; **27**: 927-939 [*RCA*] [PMID: 32628745 DOI: 10.1093/ibd/izaa167] [FullText]

99 **Attauabi M**, Wewer MD, Bendtsen F, Seidelin JB, Burisch J. Inflammatory Bowel Diseases Affect the Phenotype and Disease Course of Coexisting Immune-Mediated Inflammatory Diseases: A Systematic Review With Meta-Analysis. *Inflamm Bowel Dis* 2022; **28**: 1756-1765 [*RCA*] [PMID: 35134921 DOI: 10.1093/ibd/izac003] [FullText]

100 **Hjuler KF**, Dige A, Agnholt J, Laurberg TB, Loft AG, Møller LF, Christensen R, Iversen L. Effectiveness of interdisciplinary combined dermatology-gastroenterology-rheumatology clinical care compared to usual care in patients with immune-mediated inflammatory diseases: a parallel group, non-blinded, pragmatic randomised trial. *BMJ Open* 2021; **11**: e041871 [*RCA*] [PMID: 33910945 DOI: 10.1136/bmjopen-2020-041871] [FullText] [Full Text(PDF)]

Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: office@baishideng.com
Help Desk: https://www.f6publishing.com/helpdesk
https://www.wjgnet.com