Review Article

Gut microbiome in dermatology – A narrative review

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Abstract

The gut microbiome and human body have co-evolved in a synergistic host-microbial relationship. The ideal composition of human gut microbiota is an elusive concept, but every individual has a unique gut microbiota profile with regional differences. Newer diagnostic techniques have helped identify different bacteria and their roles in health and disease. The gut microbiome composition is affected by various factors like age, diet, immune system, environmental factors, exercise, and drugs. The microbiome has varied roles in metabolism, immune response, immune tolerance and antimicrobial protection. Diet plays an important role in maintaining the gut microbial diversity. Loss of homoeostasis in the microbiome results in dysbiosis. Dysbiosis plays a role in many dermatological diseases like atopic dermatitis, psoriasis, acne, rosacea, hidradenitis suppurativa, connective tissue disorders and many other systemic conditions like obesity, diabetes, neurological disease and malignancy. Reconstitution of the gut microbiome ecology in the form of bacteriotherapy with the reintegration of certain strains of microbiota has a beneficial role in many of these disorders.

Key words: gut microbiome, dermatology, bacteriotherapy

Science has long been intrigued by human microbiota. During evolution, microbial colonisation in animals, including humans, has resulted in a complex host-microbial relationship, influencing their genotype and phenotype. This consequently has generated a wide range of interdependent physiologic activities for their well-being.[1] The term microbiota has been used interchangeably with microbiome.[2–4] The microbiome in humans is composed of bacteria, yeast and viruses that can impact both health and diseases.[5]

The gastrointestinal tract accounts for the largest portion of the microbiome with 100 trillion microbial cells, which is thrice the number of human cells. The host-environment interface of the gastrointestinal tract has a surface area of 30 m^2, followed by the skin interface accounting for 25 m^2.[6,7]

The dynamics of the microbiome of the skin and the gastrointestinal tract appear to be interlinked. This relationship has been described by the ancient Indians and the Greeks. The Indian system of Ayurveda recognises the primary location of vata dosha in the colon. The father of modern medicine, Hippocrates, famously asserted that the gut is the basis of overall health.[8]

In 1930, dermatologists Stokes and Pillsbury first hypothesised regarding the communication of the skin and gut with the brain. They theorised that emotional states might alter the normal gut flora and contribute to systemic inflammation and diseases like acne. These findings have been recently validated and have formed the basis of the gut-skin-brain axis.[9,10]

This review provides an overview on the gut microbiome ecology, factors affecting the gut microbiome leading to dysbiosis and its role in many dermatological conditions.

Human gut ecology

One of the major challenges for scientists with respect to the gut microbiome was the inability to culture these microorganisms. However, advances in identifying

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techniques like The Human Microbiome Project of 2007, which used DNA based sequencing methods, expanded our knowledge of the human microbiome.[1] The areas of the healthy microbiome in humans include the gastrointestinal tract, skin, oral cavity, respiratory tract and vagina. Each of these sites has a unique microbial ecosystem that interacts dynamically with the host.[4,11,12]

The gut microbiome has been found to have more than 35,000 species of bacteria. These trillions of bacteria in the human colon form an extremely complex microbial ecosystem. The presence of extensive cellular and neural networks in the gut and their interactions with the other organs through chemical mediators is considered by many as the 'second brain'.[13–17]

Gut microbiota variations

There is no 'ideal and optimal' gut microbiome composition, but every human has a fingerprint-like distinctive gut microbiome profile with functions including maintenance of the gut mucosal barrier, immune tolerance, antimicrobial actions and formation of metabolites required for host nutrient metabolism.[16] The large intestine accounts for almost 70% of all the microbes in the human body.[15,18] The predominant gut phyla are Bacteroidetes, Firmicutes, Proteobacteria, Actinobacteria and more. Out of these, Firmicutes and Bacteroidetes represent 90% of the gut microbiome.[19] The Firmicutes phylum includes Lactobacillus, Staphylococcus and Streptococcus among others. Common pathogenic bacteria like Escherichia coli, Shigella and Helicobacteria belong to the Proteobacteria phylum, and Bifidobacterium and Bacterioides belong to the Actinobacteria and Bacteroidetes phylum, respectively.+

The distribution of microbiome in different parts of the gut is given in Table 1.[20,21] In addition to the spatial difference, there is an axial change in microbes from the lumen to the mucosa of the intestine [Figure 1].[15,22]

Factors affecting the gut microbiome composition

The entire gut microbiome can be divided into a stable microbiome that is highly constant over time, and an opportunistic microbiome which varies and can affect the immune system. The composition of the gut microbiome is affected by various factors like age, diet, immune system, environmental factors and exercise [Figure 2].[16,23,24]

Table 1: Distribution of gut microbiome

Stomach and duodenum	Fermicutes species like Lactobacillus, Staphylococcus, Streptococcus. Enterobacteriaceae, Helicobacter Pylori (proteobacteria phyla)
Jejunum and ileum	Lactobacillus, Streptococcus, Enterobacteriaceae, Bifidobacterium, Bacterioides
Colon	Lactobacillus, Streptococcus, Enterobacteriaceae, Bifidobacterium, Bacterioides, Clostridium, Fusobacteria
Skin	Moist areas: Staphylococcus, (Actinobacteria) Corynebacterium and Proteobacteria Oily areas: Corynebacterium, Staphylococcus Cutibacterium Dry skin: Proteobacteria

Functions of Gut microbiome

Metabolome

The larger number of genes of the gut microbiome as compared to the human genome results in immense functional superiority and versatility.[25,26] The dietary carbohydrates nourish the gut microbiome with nutrients, the gut microbiome, in turn, ferments the complex carbohydrates present in non-digestible dietary fibres and intestinal mucus. This fermentation results in the formation of short chain fatty acids like butyrate, which are energy sources for the intestinal and liver epithelial cells.[27] The various metabolomes associated with the gut microbiome are given in Figure 3.[15,18,28–30]

Antimicrobial protection

The gut microbiome plays an important role in the education and improvement of the immune system.[23] Via organism-specific microbe-associated molecular patterns such as peptidoglycan, lipopolysaccharides and bacterial nucleic acids, the gut microbiome constituents activate the pattern recognition receptor-mediated mechanisms (e.g. toll-like receptor) in the Paneth cells, which results in the synthesis of antimicrobial peptides (AMP) such as defensins, cathelicidin, calprotectin and so on. However, the gut homoeostasis can be disrupted due to overactive toll-like receptor (TLR) stimulation, leading to cytokine responses which can increase the risk of inflammatory and autoimmune disorders.[15,31,32]

Gut microbiome and diet

The food humans consume is a driving factor in the overall design and function of the gut microbiome. This microbial community interacts with the gut epithelium and immune system and maintains intestinal homoeostasis in a healthy state.[31]

Various dietary factors which affect the gut microbiome are given in Table 2.[31,33–39] There are regional variations in diet and therefore in the gut microbiome. The data from studies comparing the gut microbiome of tribes from Papua New Guinea to samples isolated from the developed areas of the United States showed higher biodiversity and lower rate for autoimmune diseases with a higher average number of bacterial taxonomic units in the two tribes when compared with the samples from United States.[17,35] Among the developed countries, the primary factor for decrease in beneficial microbes which help with fibre breakdown, is a diet rich in fat and protein and low in fibre. This can lead to an increase in pathogenic bacteria which can damage and disrupt the mucosal barrier causing systemic inflammation.[34,37]

Gut-skin axis

The gut and skin share many functionally analogous features in terms of immunological, neuroendocrinal and metabolic properties.[37] The gut-skin axis represents the sum total of

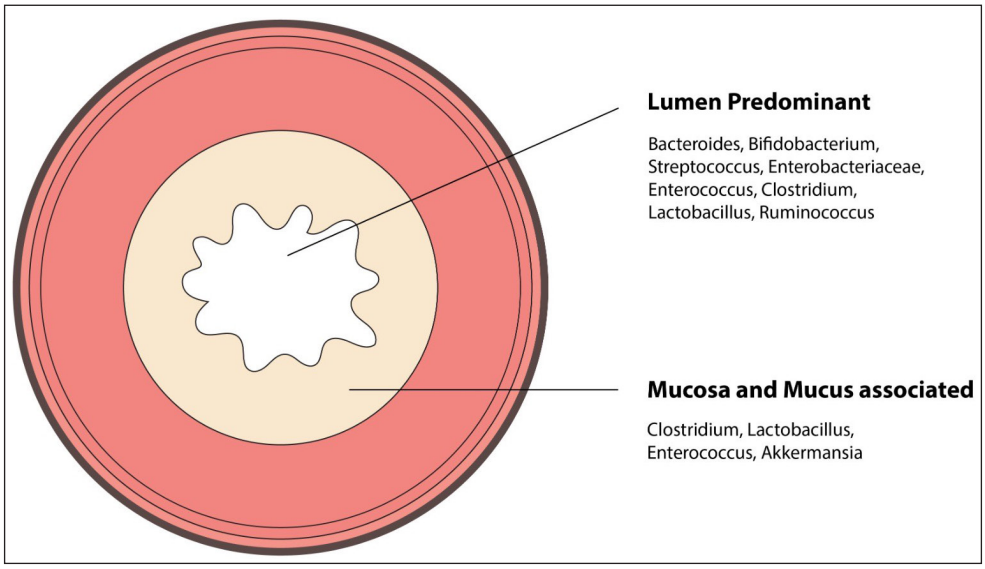

Figure 1: Luminal and mucosal microbiome.

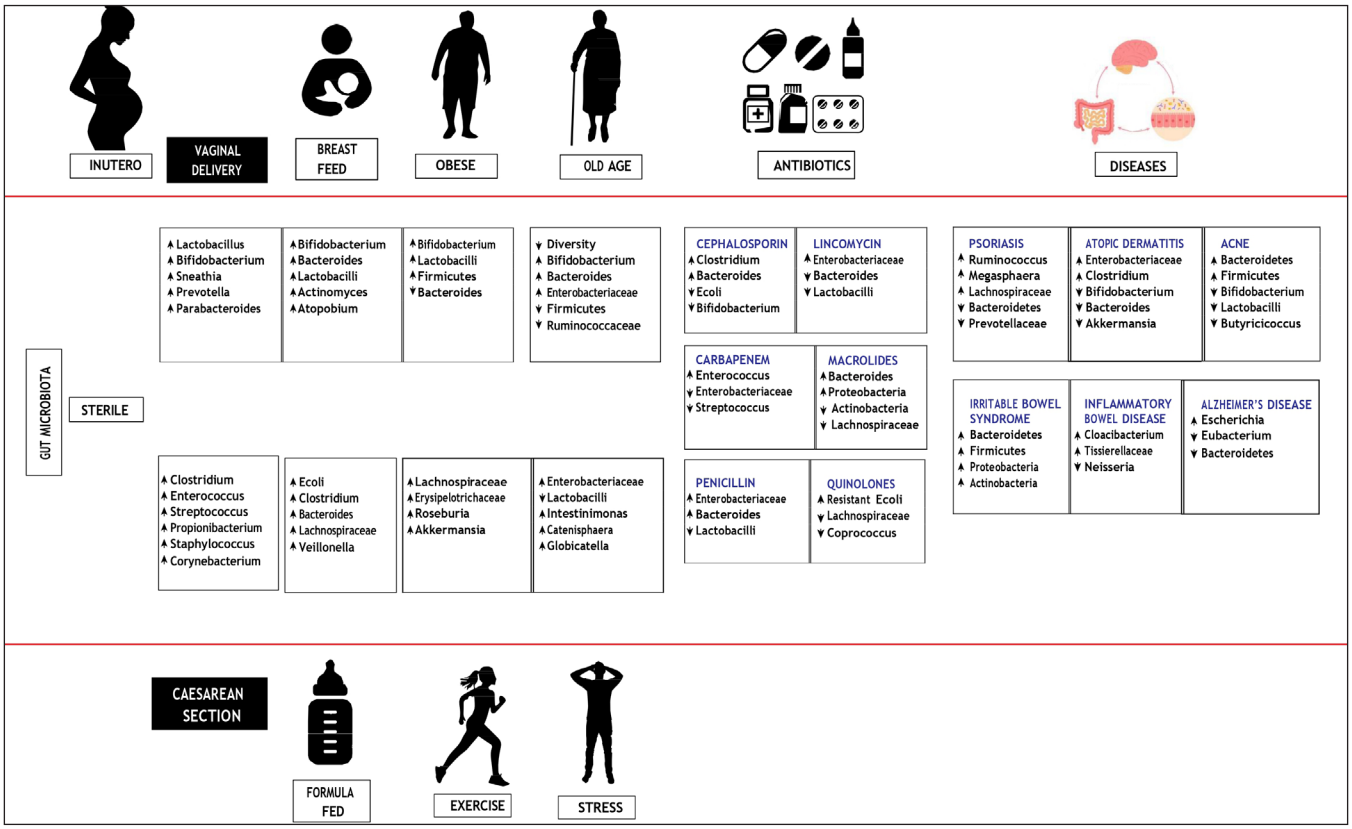

Figure 2: Factors affecting the gut microbiome composition.

the close association of the gut microbiome with skin. The concept was further expanded by Arck *et al*.[34] in 2016, who defined the concept of the gut-skin-brain axis, wherein the three organs share complex signals which modulate various interactions between the gut microbiota, emotional states and systemic and skin inflammation. Disturbances in the gut-skin-brain axis homeostasis can contribute to the development of various dermatologic, gastrointestinal and neuro-psychiatric disorders.[4,34,38] This intricate relationship requires an optimal and delicately balanced host response to prevent over utilisation of host resources by commensals while maintaining immune tolerance.[40] Any pathologic alteration in gut microbial diversity results in 'Dysbiosis'. This can make the host susceptible to many autoimmune

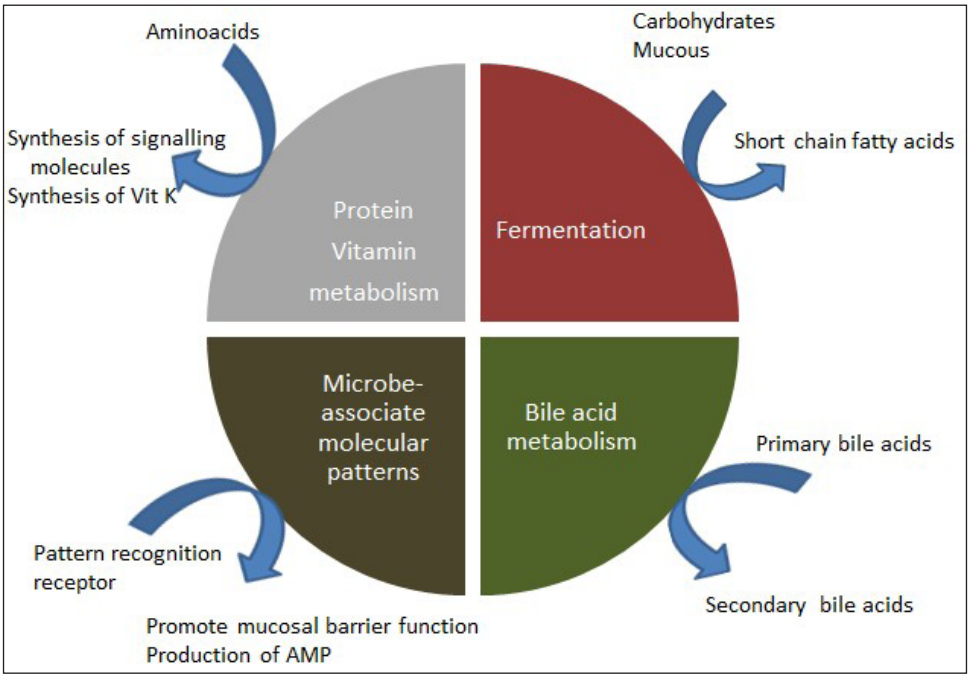

Figure 3: Metabolomes of the gut microbiome.

Table 2: Dietary factors affecting gut microbiome

Dietary fibre	Dietary fibre is the main nutrient for maintaining the diversity of the gut microbiome. Most dietary fibres are fermentable (e.g. non-digestible oligosaccharides and polysaccharides, resistant starches and chemically synthesised carbohydrates). The sources of these fibres include plant-based foods such as fruits, vegetable, nuts, cereals, legumes, nuts and tubers broadly called prebiotics.[34,35] The dietary fibres are not processed enzymatically in the small intestine but through fermentation by the gut microbiome in the large intestine to provide energy to the host. It maintains the integrity of the mucus barrier and eliminates the risk of pathogenic infection.[36–38]
Sugar	A high sugar intake induces an increase in the mucus-degrading bacteria, which decreases the expression of tight junction proteins leading to increased gut permeability and inflammation.[34]
Fats	A high-fat diet leads to reduced diversity of the gut microbiome in humans with an increased ratio of Firmicutes/Bacteroidetes. Fat-soluble vitamin D plays an important role in the regulation of gut microbiome and immune responses and has a protective role in the inflammatory bowel disease.[36]
Proteins	The quantity and source of dietary protein determines the amount and profile of bacterial metabolites. A high-protein diet shifts gut bacteria metabolism to protein fermentation and can disturb the gut mucosal homoeostasis. Plant protein has been reported to increase gut-commensal Bifidobacterium and Lactobacillus, while they additionally decrease the pathogenic Bacteroides fragilis and Clostridium perfringens and also produce useful metabolites like short chain fatty acids. High animal protein intake is associated with an increased risk of inflammatory bowel disease. Processed meat contains high amounts of sulphated amino acids with increased levels of trimethylamine-N-oxide, a proatherogenic compound that increases risk of cardiovascular disease.[36,39–42]

and inflammatory dermatoses (e.g. acne, atopic dermatitis, psoriasis, hidradenitis suppurativa, rosacea, connective tissue disorders etc.) [Figure 4].[41]

Consequences of intestinal dysbiosis

1. Inflammation – The inflammatory response of the gut epithelial membrane is systematically regulated by the epithelial cells, mucus layer, dendritic cells, natural killer cells, T-cells and macrophages [Figure 4]. Treg cells, through cytokines, like interleukin 10, transforming growth factor beta and interleukin 35, help in developing immune tolerance to self antigens and preventing autoimmune and inflammatory diseases. A balance in T helper type 1 and 2 cells activity is required for homoeostasis and preventing both chronic inflammation and allergic responses. Similarly, the T helper type17 cells, normally maintain the barrier function of the gastrointestinal tract, but when dysregulated, they contribute to the pathogenesis of diseases like psoriasis via interleukin 17 and interleukin 22.[42–44]
2. Leaky gut – Another hypothesis is that gut inflammation can increase intestinal permeability and migration of bacteria or their metabolic products from the gut into the circulation. The effect on the skin can be in the form of reduced keratin synthesis and deranged epidermal differentiation. Studies have shown the presence of deoxyribonucleic acid of the gut bacteria in the bloodstream in patients with chronic skin diseases.[9,38]
3. Neuroendocrine – The gut microbiome acts on neural pathways through the production of a variety of metabolomes, cortisol and neurotransmitters like gamma-amino butyric acid, serotonin and more. These molecules stimulate keratinocyte production of

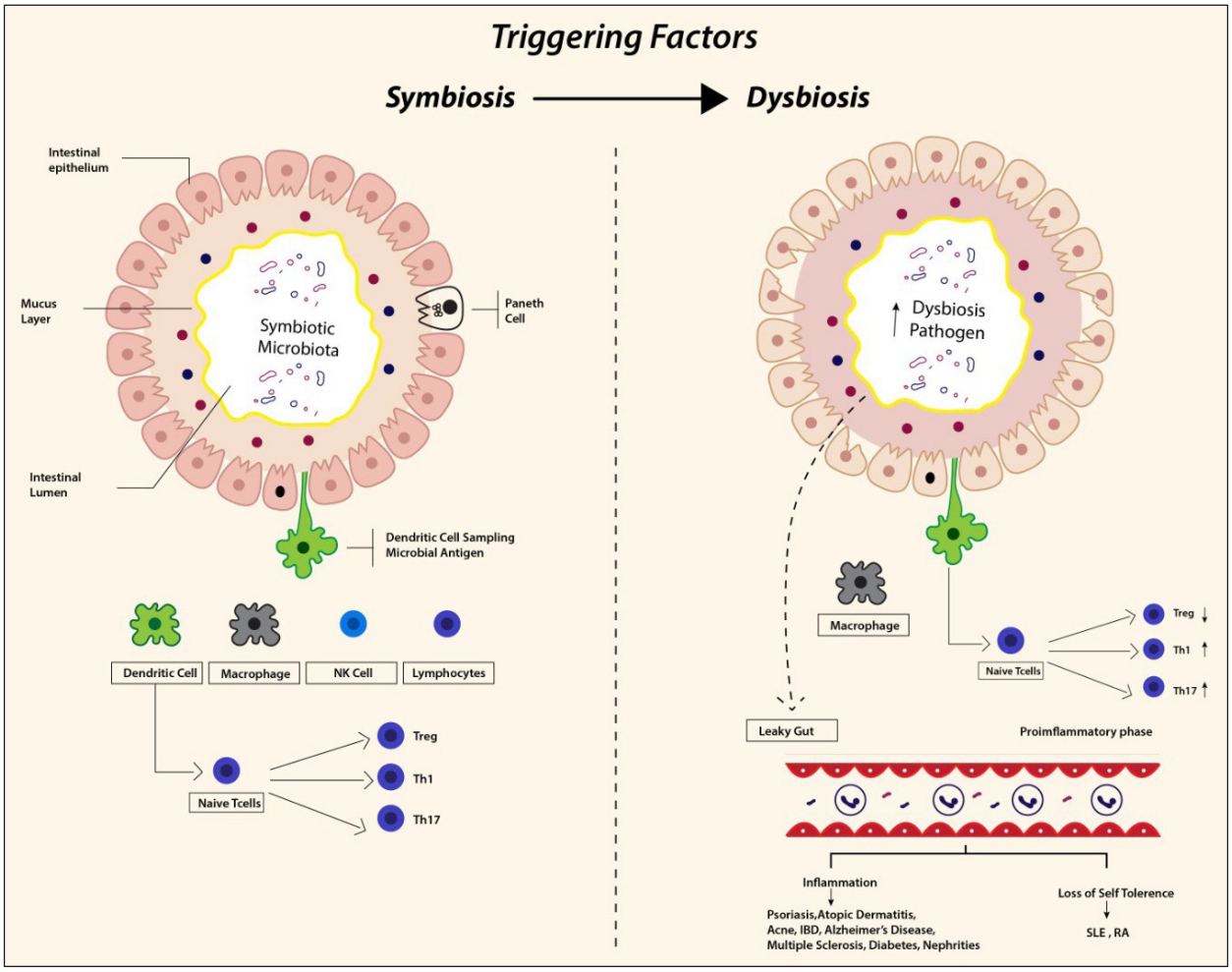

Figure 4: Immunology of symbiosis and dysbiosis. NK: natural killer cells, SLE: systemic lupus erythematosus, RA: Rheumatoid arthritis.

pro-inflammatory cytokines and enhance Langerhans cells immune responses, as seen in dermatoses like atopic dermatitis, psoriasis and rosacea.[38,45]

4. Immunological – The gut microbial ecosystem has a wide range of diverse antigens coded by more than nine million genes as compared to the human genome of around 23,000 genes. In a healthy individual, this may programme the immune system to be tolerant towards innocuous stimuli and self-antigens. However, dysbiosis may provide a fertile ground for molecular mimicry between the self and bacterial antigens, which can activate the effector immune cells to attack self-antigens, resulting in an autoimmune response.[38,46–49]

Gut microbiome and dermatoses

Recent data shows an ever-increasing association between gut microbiome and various diseases like diabetes, obesity, neurological illness, malignancy and autoimmune diseases and also many inflammatory dermatoses like psoriasis, atopic dermatitis among others.[50–55]

Atopic dermatitis (AD) – The role of the gut microbiome has been widely studied in AD. While the 'hygiene hypothesis' in AD suggests that decreased diversity and microbial exposure in early life, results in loss of immunological tolerance and an increased prevalence of allergic diseases, it is the gut microbes that form the most important component of microbial exposures.[56] The age of onset, phenotype, severity and remission of AD is associated with gut microbiome composition in the early life of an individual.[57–60] Studies have suggested that patients with AD exhibit a decreased microbial diversity and alterations in both beneficial and harmful bacteria compared to healthy subjects [Figure 2].[61–64]

Psoriasis

Psoriasis is characterised by epidermal hyperproliferation and a dermal immune response in the form of an overactive tumour necrosis factor-alpha, interleukin-17 and interleukin-22 activity. The dysbiosis in psoriasis is akin to that seen in inflammatory bowel disease with an altered Firmicutes/Bacteroidetes ratio and depletion of beneficial bacteria in psoriasis patients.[65,66]

In patients with moderate-to-severe psoriasis, markers of intestinal barrier damage such as intestinal fatty acid-binding protein are significantly elevated which may explain the underlying metabolic abnormalities in these patients.[67,68]

A recent study by Li *et al*.that used the Mendelian randomisation strategy revealed that eight specific bacterial taxa at the genus level were suggestively associated with the risk of psoriasis.[69]

Acne

The exact mechanism by which the gut microbiome is involved in the pathogenesis of acne is not established, but an immune mechanism similar to psoriasis and AD may play a role. One of the possible mechanisms may be the mammalian target of rapamycin signalling pathway. Mammalian target of rapamycin is a nutrition sensitive regulator of lipogenesis and adipogenesis. Studies show an increased expression of mammalian target of rapamycin in patients with acne as compared to healthy controls. Also, a diet high in glycaemic index was associated with greater mammalian target of rapamycin signalling, resulting in increased sebaceous activity that may trigger acne.[70–73]

The gut-skin-brain axis may also play a role due in the stress-induced aggravation of acne due to increased production of neurotransmitters by bacteria. [73–75]

Rosacea

Though the exact role of Gut microbiome (GM) in rosacea is not known, overexpression of microbe-associated molecular patterns, TLR, AMP and mammalian target of rapamycin play an important role in human innate immune protection against bacteria is seen. [41] A study evaluating the role of small intestinal bacterial overgrowth in rosacea revealed that treatment with rifaximin in these patients resulted in clinical remission of rosacea.[41,76]

Hidradenitis suppurativa (HS)

The role of gut microbiome in HS is suggested due to its shared pathogenetic mechanisms with inflammatory bowel disease (IBD) and also by the response of IBD to anti-tumour necrosis factor alpha therapies,[76,77] Different studies have implicated varied species in HS which included an increase in Escherichia, Shigella and depletion of Faecalibacterium prausnitzii.[78] HS symptoms can be aggravated by dairy products, brewer's yeast, high sugar intake, carbonated drinks and saturated fat. A plant-based or the Mediterranean diet is one of the therapeutic approaches to mitigate inflammation in HS.[79]

Connective tissue diseases

Gut dysbiosis is noted in connective tissue disorders like systemic lupus erythematosus and systemic sclerosis. The pathogenesis involves crosstalk between the commensal microbiota, immune system and metabolites. Systemic lupus erythematosus patients show a reduced Firmicutes/Bacteroidetes ratio while reduced levels of Clostridiaceae are seen in patients with scleroderma. These microbes cause an increased production of pro-inflammatory free fatty acids and a reduction in short chain fatty acids, which are anti-inflammatory.[80–82]

Vitiligo

Recent studies have shown that an altered gut microbiome may contribute to vitiligo pathogenesis. Bzioueche *et al*. compared vitiligo patients with healthy controls and found gut dysbiosis via reduced richness and bacterial species distributions in patients with vitiligo. [83,84]

Vulvovaginal candidiasis

The crosstalk of bacterial strains between the gut and vagina stimulates both local and systemic immune responses with subsequent effects on the host. The Gram-positive bacilli Lactobacillus that dominates the vaginal microbiota is believed to originate from the gut. The intestinal flora is believed to be the source of infection in recurrent bacterial vaginosis and group B streptococcal infection in pregnancy.[85,86]

Role of prebiotics, probiotics and microbiome transplant

Bacteriotherapy refers to the therapeutic administration of commensal bacteria. This has been used in the management of many inflammatory and non-inflammatory diseases. It involves the reconstitution of the microbiome ecology with the reintegration of certain beneficial strains of microbiota.[87]

Probiotic consumption is one of the most commonly practiced methods of bacteriotherapy. Probiotics have been defined as 'live microorganisms which, when administered in adequate amounts, confer a health benefit on the host'. The role of probiotics has changed from being a natural constituent of our diet to a therapeutic supplement. Consumption of probiotics induces significant colonisation by beneficial bacteria, which can effectively promote the formation of intestinal barrier while reducing allergic and inflammatory responses. Lactobacillus, Bifidobacterium and Streptococcus genera are the most commonly tested probiotics.[88]

Prebiotics refers to a 'nondigestible food ingredient that beneficially impacts the host by selectively stimulating growth and/or activity of one or a limited number of bacteria already resident in the colon, and thus helps to improve host health'.[32] These compounds are metabolised by microbiome of the colon after bypassing digestion and absorption in the upper intestine. 'Synbiotics' is a term introduced as a combination of probiotics and prebiotics.[88–90] The term postbiotic has been recently defined as 'a preparation of inanimate microorganisms and/or their components that confers a health benefit on the host'. It is composed of cell-free supernatants without metabolite specification/individualisation, cell wall components and/or intracellular compounds.[91,92]

Table 3 shows the details of studies of bacteriotherapy in dermatological conditions.[93–105] A systematic review of probiotics and prebiotics in atopic dermatitis showed a mixed

Table 3: Studies of bacteriotherapy in dermatological conditions

Author	Disease	Probiotic	Results
Gerasimov *et al.*[93]	Atopic dermatitis	Lactobacillus acidophilus Bifidobacterium lactis prebiotics	a. 33.7% decrease in the SCORAD score b. Decrease in the requirement of topical corticosteroid
Navarro-López *et al.*[94]	Atopic dermatitis	Bifidobacterium lactis Bifidobacterium longum Lactobacillus casei	a. Change of -83% in the probiotic group and -24% in the placebo group ($P < .001$) was noted
Gilli *et al.*[95]	Psoriasis	Lactobacillus rhamnosus	a. A reduction in the index of all clinical scores (Psoriasis Area and Severity Index from 4.53 ± 4.457 to 3.57 ± 3.333, body surface area from 5.44 ± 6.451 to 4.94 ± 5.961 and Dermatology Life Quality Index from 8.83 ± 8.631 to 7 ± 7.814 in the probiotic group)
Buhaș MC *et al.*[96]	Psoriasis	Probiotics (Bacillus indicus, Bacillus subtilis, Bacillus coagulans, Bacillus licheniformis and Bacillus clausii) and precision prebiotics (fructooligosaccharides, xylooligosaccharides and galactooligosaccharides)	a. Probiotic and prebiotic supplementation significantly improved quality of life b. Reduced levels of tumour necrosis factor alpha, interleukin 6 and interferon gamma and enhanced levels of interleukin 10 c. Lower levels of total cholesterol, low-density lipoprotein cholesterol , triglyceride and uric acid d. Enhancement in the diversity of gut microbiota was noted
Di Marzio[97]	Acne	Streptococcus thermophiles (topical)	a. Increase the production of ceramides when applied topically to the skin for 7 days
Jung GW *et al.*[98]	Acne	Lactobacillus acidophilus, Lactobacillus delbrueckii bulgaricus Bifidobacterium bifidum	a. Significant improvement in total lesion count four weeks
Golkar *et al.*[99]	Wound healing	Postbiotics obtained from Lactobacillus fermentum, Lactobacillus reuteri, Bacillus subtilis sp. natto	a. All three formulations containing postbiotics significantly accelerated the wound healing process b. Bacillus subtilis sp. natto cold cream manifested a better wound healing property
Rong *et al.*[100]	Pigmentation	Postbiotic supernatant of Lactobacillus helveticus (NS8-FS) NS8-fermented milk	a. NS8-FS exhibited significant radical scavenging activity b. Inhibit melanin production c. Exhibit inhibitory effects both to the enzymatic activity of tyrosinase in hairless mice and tanning guinea pig models
Catic *et al.*[101]	Wrinkles and ageing	CLS02021 is a postbiotic blend of metabolites, including organic acids, enzymes and peptides, which are a result of the co-fermentation of three proprietary probiotic strains; Lactobacillus plantarum, Lactobacillus casei and Streptococcus thermophilus was used	a. A significant difference of CLS02021 over the placebo group was observed for moisture and elasticity increase (both $p < 0.001$), pore size ($p < 0.01$) and a wrinkle depth decrease ($p < 0.05$)
Qi *et al.*[102]	Rosacea	intervention effects of probiotics and postbiotics of L. salivarius 23-006 and L. paracasei 23-008 on rosacea by constructing an LL37-induced rosacea-like mouse model	a. L. salivarius 23-006 and L. paracasei 23-008 alleviated skin lesions, reduced skin inflammatory infiltrates and decreased the expression of inflammatory factors b. Reduced the expression of cathelicidin LL37 and rosacea-associated factors by inhibiting the toll-like receptor 2/MyD88/NF-κB pathway c. Postbiotics of L. salivarius 23-006 and L. paracasei 23-008 could also ameliorate the rosacea-like phenotype in mice via the toll-like receptor 2/MyD88/NF-κB pathway, but less than probiotic therapy
Navarro-Belmonte *et al.*[103]	Alopecia areata	Lactobacillus rhamnosus Bifidobacterium longum	a. A higher proportion of Alopecia Areata patients treated with the probiotic formula showed improvement with respect to the reduction in the number of Alopecia Areata plaques (56% vs. 30%) and the affected scalp surface area (45% vs. 20%) b. Probiotic treatment led to a modification in the skin microbiota
Esmaeili *et al.*[104]	Systemic lupus erythematosus	Lactobacillus delbrueckii, Lactobacillus rhamnosus	a. ↓Inflammatory responses b. ↑Production of regulatory cells (dendritic cells) c. Live probiotics could modify properties of dendritic cells to modulatory cells, which might contribute to the induction of tolerance and renovation of immune haemostasis
Liu *et al.*[105]	Atopic dermatitis	Faecal microbiota transplant once a week for three weeks	a. Improvements in Eczema Area and Severity Index scores b. Decreased the helper T (Th2 and Th17) cell proportions and the levels of tumour necrosis factor alpha and total immunoglobulin E in serum

SCORAD: SCORing Atopic Dermatitis

response with many studies showing a lower SCORAD score following consumption of oral probiotics, while few studies showed moderate or no response. Multistrain synbiotics and vitamin D3 supplements in addition to regular treatments have also shown to be effective in reducing severity of AD in infants.[94,106,107]

A Delphi Consensus Statement on the Role of Probiotics in the treatment of AD noted that probiotic supplementation for 8–12 weeks should be a part of the complementary therapy in the management of AD and associated flare-ups. Probiotic therapy may also serve as a strategy to reduce steroid usage or maintenance therapy in high-risk cases with frequent flares.[108]

There are few clinical trials of probiotic use in psoriasis. Probiotics and prebiotic supplementation in patients with psoriasis on therapy resulted in better psoriasis score, decreased inflammatory markers and skin thickness compared to those not receiving supplementation.[96,109]

Probiotic supplementation in acne is known to suppress inflammation through their antibacterial and anti-inflammatory effects. Probiotics may also lower the glycaemic load, reducing mammalian target of rapamycin and insulin-like growth factor-1 signaling, and therefore decreasing sebaceous gland hyperplasia and keratinocyte proliferation in acne.[110,111]

A Cochrane review on the use of probiotics in non-pregnant women with vulvovaginal candidiasis as an adjuvant therapy found an increased rate of short-term clinical and mycological cure and decreased relapse rate at one month, but this did not translate into a higher frequency of long-term clinical or mycological cure.[112]

Microbiome transplantation has been used for a variety of diseases like ulcerative colitis, Crohn's disease, recalcitrant Clostridioides difficile infection, bacterial vaginosis, recurrent hepatic encephalopathy, milk allergy, autism spectrum disorder and intractable melanoma. While the concept of microbiome transplantation was earlier synonymous with faecal microbiota transplantation, it has now expanded to encompass the skin and vaginal microbiome for the treatment of disorders related to these body sites.[113–119] Faecal microbiota transplant introduces a healthy donor's gut microbiome to a patient with underlying disease.[87] It can be delivered to the recipient by rectal enemas, endoscopy, tube insertion or oral capsules via colonoscopy or endoscopy. It is important in faecal microbiota transplant that the stool donor should have good general and bowel health without any detectable infectious agents.[117] It has been tried in patients with atopic dermatitis, alopecia universalis, psoriasis and psoriatic arthritis with varied responses.[120–122]

Currently, the overall clinical efficacy due to bacteriotherapy appears to be variable and contradictory, with many studies showing limited evidence, while some systematic reviews and meta-analyses have shown strain and region-specific efficacy. These disparities may be due to the lower power of studies and strain variability between studies. In the future, a clearer curated database of microbial colonies in various diseases will enable appropriate donor-recipient combinations. Also, a deeper understanding of the mechanisms involved in the microbiome composition and activity post-therapy will enable the application of bacteriotherapy in an individualised manner.[117]

Conclusion

The gut microbiome and the human body are intricately woven and synergistically related as a part of the evolutionary process. The gut microbiome is very unique to an individual with many beneficial functions in a homoeostatic state. Dysbiosis of the microbiome appears to play a role in many diseases, including inflammatory diseases and autoimmune disorders. Recent studies have recognised the role of the gut microbiome in various dermatoses, revealing pathogenic patterns analogous to other inflammatory conditions and identifying novel mechanisms linking the gut to skin health. Understanding the interplay between the gut microbiome and diverse dermatological factors responsible for diseases, along with targeted modulation of the microbiome, presents a promising avenue for alleviating disease processes.

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