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Gut Health & Glow-An Overview of Enterodermatic Symbiosis

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Abstract

Skin and gut are the largest interfaces that harbor microorganisms in varied shapes and sizes. This microbiome has a significant symbiosis that can be affected by genetics, host factors, environmental factors, exogenous compounds, diet and dermatological diseases which alter the function as well as structure. This causes a profound impact on one's health. The gut-skin axis equilibrium is maintained by the immune mechanisms that govern the process. However, once this balance is disturbed leading to dysbiosis, several toxins accumulate leading to systemic inflammation that gives birth to dreadful diseases. Hence understanding the pathophysiological mechanisms associated with this relationship can assist in discovering novel treatments that would strategically prove to be beneficial in maintain a healthy and balanced life.

Keywords: microbiota; inflammation; gut; skin

Introduction

Skin is the largest organ of our body and provides the epithelial surface for the interaction of millions of microbes [1]. It serves a multitude of functions including protection, lubrication as well as secretion of essential elements that are vital not only for its health but also for maintaining its smoothness and shine. Just like skin, gut also assists in one's overall wellbeing. It is also one of the largest interfaces (30 m^2) between the host and its environment [2]. Despite serving just as an organ of digestion and absorption, it secretes substances and enzymes & keeps the immune system on check. The uncontrolled growth of harmful microorganisms is prevented by the first line defense & barrier functions of gut. Both the gut and skin are immensely packed with microbiota. It is estimated that the skin has about 10^{12} microbial cells while the gut accounts for 10^{14} microbial cells. Culture-independent methods have revealed that healthy human skin harbors >1,000 bacterial species, mainly within the genera Brevibacterium, Propionibacterium, Micrococcus, Staphylococcus, Streptococcus, Corynebacterium and Malassezia genus [3,4]. The microbiome benefits the host by shaping the immune system, protecting against pathogens, disintegrate down metabolites, and maintain a healthy barrier [5]. Researchers have hypothesized that increased microbial diversity in the gut confers resilience, thereby promoting health and preventing disease [6]. The gut-skin window is what we reflect inside out. The symbiosis can be disturbed by various genetic and environmental factors that play a crucial role in maintaining one's health. Harboring the good commensals and eliminating the bad friends is served by a perfect skin-gut relationship. This review focuses on the symbiotic importance of skin-gut in establishing a healthy wellbeing.

Microbiota

One of the essential, yet under looked and underestimated companion of the skin health is the "gut microbiota". It is also referred to as a "superorganism" or "the last unexplored human organ," or an invisible organ [7]. The gut harbours the highest number of microbes in vibrant shapes and sizes. This bio community which includes virus, bacteria, fungi, protozoans along with their genetic material is referred to as gut microbiome. The most popular phyla included Bacteroides, Firmicutes, Actinobacteria, Proteobacteria, Verrucomicrobia, Fusobacteria, Tenericutes, Spirochaetes, Cyanobacteria and Sacchari bacteria. The prevalent fungal genera include Saccharomyces, Candida and Cladosporium [8,9]. Among Archaea, the predominant genera in the human gut are Methanobrevibacter, Methanosphaera, Nitrososphaera, Thermogynomonas viruses and phages, on the other hand, can act as reservoirs of genetic material in the gut and destroy microbial cells [10]. This microbiome is not always same and is modifiable according to the age and development of

the individual. It can also vary according to regional sites present in the skin such as moist over axilla, antecubital fossa & umbilicus and dry over buttock, palms while oily at sites such as glabella, alar crease, manubrium and occiput. Dietary factors, genetic and environmental factors also have a significant impact on its variability. A clear consensus as to how they affect our metabolism and personality is yet to be explored.

Good Versus Bad Microbiota

Healthy microbiota

The skin microbiota is essential for the integrity of the skin's natural barrier. The barrier is colonized by either resident or transient flora. Resident microorganisms are the core microbiota that are stably colonized on the skin despite exposure and are harmless to the host. They add some or the other benefits to the host [11] while transiently living flora from the external environment can persists for hours to days before disappearing Under normal conditions, the transient skin microbiota also appears to be nonpathogenic [12]. Apart from acting as barrier, good microbiota protects against potential pathogens by AMP production. They activate keratinocyte aryl hydrocarbon receptor [13], immune system regulation [14], wound repair [14,15] and protection against skin cancer [16]. For example, Certain strains of *Staphylococcus epidermidis*, skin commensal, benefit the host by activating the host innate immune response against pathogens via toll-like receptor (TLR) 2, producing AMPs and phenol-soluble modulins (PSMs) against pathogens, such as *S. aureus* and *Streptococcus* spp., and stimulating the production of AMP from the host [17]. Microbial protease enzymes are involved in the desquamation process in the stratum corneum. Proteases from *S. epidermidis* inhibit biofilm formation and eliminate colonization by pathogenic bacteria [18], accelerates wound closure by recruiting neutrophils and activating type I interferon (IFN)-producing plasmacytoid dendritic cells [19]. Other skin commensals, such as *Staphylococcuslugdunensis* and *Staphylococcus hominis*, also inhibit *S. aureus* growth through the production of the antibiotic lugdunin and lantibiotics, respectively [20].

Pathogenic Microbiota

The Balanced interplay between the host and bacterial population is continuously affected by endogenous factors such as genetics, hormones and aging or exogenous factors such as lifestyle, geographical location or usage of antibiotics that alter the composition of the microbial inhabitants and host's skin barrier function Therefore the disease progression in several inflammatory diseases varies with the altered composition of cutaneous microbiotome. Using genomic and metagenomic approaches, the roles of microbial components that stimulate or modulate host responses have been proposed. For example, Staphylococcus aureus, a pathogenic skin microbiota is responsible for several skin conditions such as atopic or eczematous dermatitis, skin & soft tissue infections (SSTI), endocarditis, Toxic shock syndrome and osteomyelitis. However, this opportunistic pathogen, asymptomatically colonizes more than 30% of healthy individuals in relatively low and barely detectable abundance levels under steady state [21], indicating that additional host or environmental factors or potentially pathogenic bacteria are required to elicit skin inflammation. Similarly, certain strains of *S. epidermidis* opportunistically demonstrate pathogenicity even though they potentially benefit the host [22]. Certain strains of *C. accolens* inhibit the growth of *Streptococcus pneumoniae*, a potentially pathogenic bacterium that causes pneumonia, septicemia, and meningitis [23]. These findings suggest that skin microbiota is beneficial to the host in most aspects but can also exhibit pathogenicity by causing under certain circumstances.

Pathophysiological Mechanisms Pertaining to Immunobiology of Gut & Skin

The inner surface of the gut and the outer surface of the skin, both are covered by epithelial cells (ECs) which have direct contact with the exogenous environment [24]. This way, the immune system is continuously trained to distinguish between harmful and beneficial compounds. The natural surfaces of the skin and gut and the released substances offer some of the excellent first line barrier defenses that prevent the entry of microorganisms much before the pathogen is actually handled by the immune systems.

First Line defenses

1) Keratin which is present in skin's outermost stratified squamous epithelium provides a formidable physical barrier to most microorganisms and makes the skin resistant to weak acids and bases, bacterial enzymes, and toxins [25,26].

2) Mucosae – Another barrier that comprises a glycoprotein layer on top of the epithelium provides a residence to the commensal bacteria. The epithelial

membranes also produce protective chemicals that assist to eliminate microorganisms.

3)Acidity of skin ranges from pH of 5.4 to 5.9 inhibits bacterial growth and creates a hostile environment for potential pathogens and [27].

4)Sebum produced by the sebaceous glands acts as a seal for hair follicles and contains several antimicrobial molecules as well as specific nutritional lipids for beneficial microorganisms [28,29]

Similar to skin, the gut also provides natural barriers against the invading pathogens.

5) Saliva and lacrimal fluid in the digestive tract contain lysozyme, sAcid and protein digesting enzymes of the stomach mucosae

6) Mucus secreting cells traps microorganisms that enter the digestive and respiratory tract [30,31].

Second line defenses

The second line of defense are the antimicrobial peptides (AMPs), phagocytes, and innate lymphoid cells (ILCs) [32]. AMPs, such as α- and β- defensins, are secreted by localized immune cell types such as macrophages, T cells, B cells and mast cells (MC). Furthermore, Mast cells can produce the antimicrobial peptide, cathelicidin and contribute to microbiome-tissue homeostasis in the dermis. Microbial signals are sensed by RORgt group 3 innate lymphoid Cells that produce IL-17 and IL-22. The latter acts directly on the intestinal epithelial cells (IECs) and activates damage repair mechanisms, AMPs and mucin genes [33]. Pathogens can directly bind to the TLRs tors (NLRs), and (RIG-I)-like receptors (RLRs), (NOD)-like and activate complement receptors of MCs, subsequently releasing inflammatory mediators, which aid in microbial immune responses [33]. By expression of co-stimulatory molecules and secretion of inflammatory cytokines, TLR4 elicits innate response. AMPs produced by keratinocytes, such as cathelicidin and psorasin, provide an effective barrier function to the skin [34,35]. Similar to skin, immune cells are also present inside the gastrointestinal tract. The digestive tract is lined by a single layer goblet cell (mucus secretion), enteroendocrine cells (hormone secretion), enterocytes or colonocytes [36]. while the Paneth cells that are enriched in the crypts of the small intestine, secrete AMPs, which integrate in the complex mucus layer [37].

Microbial-associated molecular patterns (MAMPs) are sampled through antigen uptake by membranous (M) cells and goblet cells to dendritic cells (DCs), together with direct transepithelial luminal DCs. Plasma cells in Peyer's patches, which are stimulated by dendritic cells, produce IgA in the lamina propria in a T cell-independent manner [38]. The control of immunological proces within mucosal tissues is dependent on the interaction between epithelial cells and dendritic cells, as both cell types are involved in the sensing and sampling of antigens.

Innate immune cells of GALT

The gut mucosal defense is controlled by GALTs. These are comprised of microfold cells (M cells), conventional lymphocytes (regulatory T cells (Tregs), helper T cells (Th cells), cytotoxic T lymphocytes, IgA producing B cells) and professional phagocytes (DCs, mast cells, neutrophils, and macrophages) and unconventional lymphocytes, such as innate lymphoid cells (ILCs) and mucosal-associated invariant T (MAIT) cells. The M cells evolve through the process of phagocytose and transcytose to extrude out the harmful compounds or antigens across mucosal barrier to lamina propria. Secretory IgA controls the inflammatory responses against the gut microbes by spatially dissociating the host tissue and gut microbes. The commensal bacteria-specific lymphocytes accumulate in Peyer's patches and lamina propria of the gut, which shapes the gut microbial profile toward homeostatic balance. Defensins act against bacteria by generating pores in their membranes while Cathelicidins help to keep the epithelial barrier intact. Dendritic cells establish the specificity of CD4+ Th17 cells toward commensal microbes as the result of presentation of antigen to Major histocompatibility complex II (MHCII).The CD4+ Th17 cells produce the cytokine Interleukin 22 (IL-22), which enhances secretion of host AMPs.[39] The integrity of the intestinal barrier along with the action of mucus, immune cells, IgA, and antimicrobial peptides (AMPs) produced by epithelial cells prevents the entrance of gut bacteria into the bloodstream, ultimately maintaining skin homeostasis.

Gut And Skin Microbiome Synergy

The homeostasis of the skin is largely affected by the interaction of microbiota with the immune system. The Gut's microbial inhabitants have a significant effect on human body and can assist in multitude of metabolic and immune functions. The catabolic end products obtained from the fermentation of complex carbohydrates and other undigested food components by the intestinal microbes are incorporated into the body's short chain fatty acids

(SCFAs). Therefore, any alteration in the gut microbiota's composition or metabolic activity may also alter fatty acid levels [40]. Microbes synthesize bile acids, protect against pathogens, fortify the immune system, and impact bone development through Short Chain Fatty acids (SCFA) [41]. SCFAs help maintain the integrity of the blood-brain barrier and increase the epithelial barrier function of the skin and combat inflammation. In colon, SCFAs also promote the development of Tregs, dendritic precursor cells precursors and IL-10 production. SCFAs also modulate the immune system and inflammatory response and affect lipid and glucose metabolism [42]. Also, certain products that are produced by microbes can affect neurological function, mood, and behavior. The microbial commensals metabolize indigestible complex polysaccharides into essential nutrients such as vitamin K and B12, butyrate, and propionate [43,44]. that have a positive effect on the epithelial barrier integrity.

In fact, these byproducts may be anti-tumorigenic, as SCFAs, butyrate, acetate, and propionate, produced by the fermentation of dietary fibers by colonic microbes, have also been shown to induce apoptosis in colorectal tumor cells [45]. Propionic acid promotes skin homeostasis by reducing inflammation by antimicrobial effect while Sodium butyrate is useful in treatment of hyperproliferative skin diseases through modulation of several key cellular processes including differentiation, proliferation, and apoptosis. Gut microbiota may also convert excess proteins and amino acids into certain toxins, like indoxyl sulfate, trimethylamine N-oxide (TMAO), and p-cresyl sulfate, which may be involved in a number of diseases [46]. Apart from this, the gut microbiome serves as the largest endocrine organ that has the ability to produce atleast 30 hormone-like compounds like short chain fatty acids (SCFAs) cortisol and neurotransmitters. The neurotransmitter tryptophan plays a key role in regulating skin inflammation and activation of AhR and inhibition of TSLP production in keratinocytes. Dopamine has a causal role in Inhibition of hair growth by stimulation of catagen phase. The inhibitory neurotransmitter GABA plays the control mechanism by inhibiting neurons responsible for itch signals in the spinal cord.

Skin Gut Connection Channel

The "brain-gut-skin axis", was initially proposed by John H. Stokes and Donald M. Pillsbury in 1930 [47]. There are several suggested mechanisms and theories that elucidate the connections of gut and skin. Although no clear proven consensus about the surety is obtained and requires further research. The authors suggested that increased epithelial permeability in the gut triggers T-cell activation and disrupts immunosuppressive cytokines and T regulatory cells responsible for establishing tolerance, leading to systemic inflammation that can disrupt cutaneous homeostasis [48]. Another theory suggests that an increased intestinal permeability associated with altered gut flora allows for direct migration of inflammatory products into the systemic circulation. The hormone-like pleiotropic compounds that are produced by the gut microbiome are released into the bloodstream and can act at distant organs and systems like skin. Additionally, it has been suggested that altered levels of neurotransmitters, such as acetylcholine, norepinephrine, and dopamine produced by gut organisms can communicate with peripheral organs through neuronal pathways not yet identified [49]. However Current evidence suggests that it is likely due to a combination of both neurologic and immunologic responses to environmental shifts, resulting in chronic systemic inflammation that can ultimately affect the skin.

Gut Dysbiosis

The term dysbiosis refers to any alteration in the composition or functionality of the gut microbiota compared to what is considered healthy, thereby disrupting the delicate balance that exists between skin and the gut. It characterized by a less diverse and less stable microbiota, while the pathogenic and harmful bacteria may increase. It can impair both local and systemic immune responses, weaken the protective barriers in our mucosal tissues, and disrupt the signaling of cytokines [50]. Dysbiosis impairs skin cell's natural ability to differentiate, rendering the bacteria to enter the bloodstream, accumulate in the skin leading to reduced skin hydration and impaired keratinization. In addition, intestinal dysbiosis can make the intestinal lining more permeable, which can cause an imbalance between regulatory T-cells and the activation of effector T-cells which in turn, initiate a cycle of chronic systemic inflammation [51]. Gut dysbiosis is considered to contain biomarkers such as free phenol, p-cresol, and aromatic amino acid derivatives that are synthesized by a disturbed equillibirium [52]. Gut microbiota may also convert excess proteins and amino acids into certain toxins, like indoxyl sulfate, trimethylamine N-oxide (TMAO), and p-cresyl sulfate, which may be involved in a

number of diseases [53]. Recent research has shown that modifications in gut microbiota can lead to the development of numerous dermatological conditions such as atopic dermatitis, psoriasis, acne vulgaris. Other less common but concerning disorders such as rosacea, hidradenitis suppurativa, and chronic spontaneous urticarial, erythema nodosum, and pyoderma gangrenosum [54,55]. can also occur. Moreover, dysbiosis can also influence one's susceptibility to allergic diseases and asthma.

Factors Affecting Gut Microbiome and Skin Axis & Healthy Recommendations

Since longtime it is known that a healthy dietary lifestyle is essential for a maintaining a healthy body. Dietary habits have a significant impact on the health particularly the gut. Many dermatological conditions are affected by changes in the dietary patterns of the individuals via an altered microbiome. The influence can be both positive or negative [56]. Several drugs that are most commonly used as antibiotics for infections are found to be the one of the culprits in disrupting the growth of good bacteria. Although acting against infectious and pathogenic organisms, the antinflammatory and immunomodulatory properties can also be used in the treatment of various dermatological diseases such as acne, rosacea, pyodermas etc. However unprecedented and accelerated use of antibiotics can cause antibiotic resistance as well as promote the growth of pathogenic organisms. For example, the use of antimicrobials such as levofloxacin and moxifloxacin are associated with significantly increased numbers of Candida species in the human gut. The increased colonization of which is associated with erythema and accelerated ageing [57].

Some Healthy Alternatives to Increase Microbiome Reserve Include

High protein High fibre diet

Ingestion of diet rich in high collagen peptide such as the one containing whey and pea protein extracts increases the abundance of gut commensal genera such as Lactobacillus and Bifidobacterium while simultaneously decreasing the presence of pathogenic organisms such as Bacteroides fragilis and Clostridium perfringens. Also, SCFA levels in the intestinal mucosa are increased with the consumption of pea proteins, which are critical for keeping the mucosal barrier intact [58]. A high-collagen peptide diet contains high quantities of microbes, can protect the skin from aging and promote wound healing [59].

Pre and Probiotics

International Scientific Association for Probiotics and Prebiotics (ISAPP) declares that the substrates that upon utilization by microorganisms provide useful properties to the host are prebiotics [60]. They also promote the flourishment and growth of probiotic bacteria and facilitate a smooth equilibrium between skin and gut [61]. Hence incorporation of these substances is an essential step towards a healthy skin. Prebiotics namely galactooligosaccharides, inulin, fructooligosaccharides, polydextrose, lactulose, sorbitol and xylitol modulate the gut microbiome and are healthy alternatives for skin [62]. The galactooligosaccharides (GOS), are one of the major components of breast milk that can be fermented into SCFAs by members in the genus *Bifidobacterium.* Furthermore, GOSs have been used in the treatment of photoaging diseases for decades [63]. While human milk fatty contacting palmitate plays a prebiotic effect on the gut microbiome of infants by positively influencing the abundance of *Bifidobacterium* spp. and *Lactobacillus* spp [64]. Probiotics are defined as "live microorganisms which when administered in adequate amounts confer a health benefit to the host" [65]. They prevent colonization of gut by pathogens and at the same time provide anti-inflammatory responses by producing metabolites that embibe anti-inflammatory properties. The most common probiotic microbes that are currently in use belong to the genera *Bacillus, Bifidobacterium, Enterococcus, Escherichia, Lactobacillus, Saccharomyces*, and *Streptococcus* [66].

The beneficial role of probiotics in prevention and control of dermatological diseases can never be over looked. For example, administration of *E. coli* strain Nissle improved skin health in patients with acne vulgaris [67]. Strains of *Lactobacillus paracasei* was found to improve skin sensitivity and restoration of skin barrier function after daily oral administration [68]. While addition of *Lactobacillus reuteri* to drinking water resulted in improved epidermal thickness, increased folliculogenesis, lower skin pH, and enhanced production of sebum-producing epithelial cells [69]. Dietary fibres, the role of whom, can never be underemphasized. Though Dietary fiber cannot be digested by the human body, it is mainly metabolized by the microbiome in the colon. The metabolism of dietary fibres promotes the growth of essential bacteria in the body and increases the populations of Bifidobacteria and the Lactobacillus/Enterococcus group [70]. Foods containing complex dietary

carbohydrates can be converted into short chain fatty acids, including propionate, acetate and butyrate through fermentation by the gut microbiome. Gut commensal microorganisms influence mucosal immunity through the development of Tregs within the colon, which is mediated through short chain fatty acids (SCFAs) [71]. For example, propionic acid, which is formed during the fermentation of dietary fiber by the *Propionibacterium* genus, is antibacterial and can kill the most prevalent community-acquired methicillin-resistant *Staphylococcus aureus* strains USA300 [72].

Short Chain Fatty acids (SCFAs) derived from fiber via the gut can also influence the prevalence of certain skin microbial groups that subsequently affect cutaneous immune defense mechanisms. SCFAs play a role in the expression of Foxp3 (Forkhead box protein P3) that helps in regulating the development and function of Tregs, thus improving the regulatory T cell function. By increasing epithelial barrier function and inducing various anti-inflammatory effects, they strengthen the gut's function and integrity [73] modulate respiratory diseases and prevent the development of inflammatory disorders, like allergy, arthritis, and colitis [74]. SCFA's regulate the metabolism of lipids and glucose, and inhibit the buildup of potentially harmful metabolic by-products, such as D-lactate [75].

High-Low fat diet

Diet rich in High-fat are responsible for reduction in mucus layer thickness, gut microbial diversity and induction of higher concentrations of lipopolysaccharides. This leads to a loss of colonic epithelial integrity and barrier function and an increase in the release of pro-inflammatory cytokines, all of which adds to further systemic inflammation [76]. Refined and recooked hydrogenated oils such as vegetable oils, soybean, sunflower, safflower corn can cause inflammation of the gut. This in turn spreads the cycle of events to the body and shows up on the skin too. Skin wound healing may also be delayed by a high-fat diet and alcohol, which exacerbate the inflammation of the skin and oxidative stress [77]. A diet high in industrial trans-fatty acids increases the abundance of harmful microbes such as Desulfovibrionaceae and Proteobacteria while suppressing populations of advantageous microorganisms like Bacteroidetes, Lachnospiraceae, and Bacteroidales [78]. In addition to this, supplementation of foods rich omega-3 polyunsaturated fatty acids also increases SCFA-producing bacteria [79].

Skin-Gut Microbiome and Dermatoses

Skin plays an essential role in maintaining homeostasis by working in harmony with the gut. It helps in maintain hydration and regulate temperature balance of the body. The gut microbiome influences the signaling processes that maintain epidermal differentiation and transcription. These steps are crucial for skin renewal and regeneration. However, once this symbiosis is altered due to external and internal factors, it can pave way to several infectious and noninfectious inflammatory dermatoses [Table 1].

Dermatoses	Associated gut microbiota	Beneficial effect	Pathogenic organism at risk	Mechanism of action	Effects
Atopic dermatitis	Bifidobacterium animalis subsp. lactis [LKM512 Bacteroides thetaiotaomicron	Reduce the scratching behavior in atopic dermatitis Anti-inflammatory action	Faecalibacterium prausnitzii Overload of Clostridium difficile and Escherichia coli *Akkermansia muciniphila* in infants with AD is	Chronic atopic dermatitis progression resulting in gut epithelial barrier impairment Immune dysregulation as a result of decreased Treg cell inducing beneficial bacteria associated with intestinal barrier dysfunction and skin lesion deterioration	Dysregulation of gut epithelial inflammation Early onset of atopic dermatitis
Acne	1)Lactobacillus casei & paracasei 2)Firmicutes Lactobacillus plantarum, L. rhamnosus, 3)Lactobacillus bulgaricus, and	Reducing the severity of acne vulgaris by selectively lowering TGs in skin surface lipids.	Cutibacterium acnes. Bacteroides Akkermansia,	1)Inhibition of mast cell degranulation, TNF-α release, edema and vasodilation, and thereby speeding up the restoration of barrier function 2) Dysbiosis by altering the serological cytokine levels promoting inflammation	Reduce the size of acne lesions as well as inflammation

	Streptococcus thermophiles				
Psoriasis	Faecalibacterium prausnitzii, Akkermansia muciniphila and Ruminoccocus Bifidobacterium infantis	Prevention of colonization of pathogenic flora on skin by competitive inhibition and the SCFAs production decreased plasma levels of TNF-α, IL- 6, and C-reactive protein (CRP)	Higher abundance of Staphylococcus and Streptococcus		
Rosacea	Peptococcaceae and Methanobrevibacter		Helicobacter pylori Acidaminococcus and Megasphaera increase	Production of cytotoxin and proliferation of reactive oxygen species like nitric oxide leading to vasodilatation,inflammation and immunomodulation	Small intestine bacterial overgrowth
Wounds	*Lactobacillus fermentum, Lactobacillus reuteri, and Bacillus subtilis*				
Alopecia areata	*Lactobacillus kunkei* L. lactis strain H61	Improving hair follicles and skin hydration			
Hidradenitis suppurativa			*Ruminococcus gnavus* and *Clostridium ramosum.*		

Atopic Dermatitis

It is an inflammatory skin disorder that can present as a bimodal peak. The disease is characterised by dry, itchy rash with a vicious itch-scratch cycle. Atopic dermatitis affects up to 20% of infants and 3% of adults worldwide and is often associated with other diseases like allergic rhinitis and asthma [80]. Infants with a dysbiosis of the healthy microbiome can present with an early onset atopic dermatitis. The same was demonstrated by Zheng et al [81]. that abundance of *Akkermansia muciniphila* in infants with AD is associated with intestinal barrier dysfunction deterioration of skin lesions. Diet plays a very crucial role in the progression and maintenance of a healthy gut microbiome. In the urbanized and overhygienic society the "Hygiene hypothesis" holds good. It states that over development of hygiene in western diets can lead to development of numerous immune mediated inflammatory diseases such as atopic dermatitis, psoriasis etc due to under stimulation of microbial mileu. The mechanism of the Western diet or high-fat diet (HFD) relies on the resulting intestinal dysbiosis, leading to an increase in the ratio of Firmicutes to Bacteroides leading to an atopic Th2-skewed response. Upon activation and recruitment Th2s substantially produce inflammatory cytokines like IL-4, IL-5, and IL-1 that ultimately cause an enhanced production of IgE [82]. According to a study, dysbiosis of *F. prausnitzii* in patients with Atopic dermatitis was associated with an increased expression of a variety of nutrients like the mucin components like GalNAc and L-fucose that are released from damaged gut epithelium. The leaky gut and dysregulated inflammation of the gut epithelium causes an increased gut permeability due to damaged gut epithelium and causes the leakage of toxins along with food residues and pathogens to gain an access to the blood circulation.

Acne

Acne is a disorder of sebaceous glands characterized by formation of comedones, papules or pustules. According to recent studies acne is associated with significant reduction in the prevalence of *Actinobacteria*, *Bifidobacterium*, *Butyricicoccus*,

Coprobacillus, and *Lactobacillus* species along with an increased abundance of Proteobacteria [83]. According to a hypothesis, sterol regulatory element-binding protein 1 (SREBP-1), sebum fatty acid, and sebum triglyceride stimulated by nutrient signaling disruption can lead to flourishment of *Propionibacterium acnes*.[84] Other factors like diversity of the C. acnes population, porphyrin production, mobile genetics Elements, associated CRISPR/CAS loci and production of SCFAs can also contribute. Various metabolic pathways also influence the pathophysiology of acne vulgaris, such as the mTOR pathway that becomes activated by high glycemic load which in turn activates the IGF-1 and FOXP-1 expression. This leads to increased insulin/insulin-like growth factor (IGF-1) signaling and enhancing the cytoplasmic expression of FoxO1 (Forkhead box transcription factor O1) that stimulates the mammalian target of rapamycin complex 1 (mTORC1), which ultimately leads to acne development [85]. The mTOR-mediated activities such as cell proliferation and lipid metabolism are controlled by metabolites produced by gut microbiota. Hence disruptions in the gut barrier and dysbiosis can create a positive feedback cycle of metabolic inflammation, contributing to the underlying processes of acne. Another member known as neuropeptide Substance P is implicated in acne and intestinal dysbiosis, can trigger inflammatory signals that contribute to acne development. Several factors pertaining to gut such as Gastrointestinal dysfunction associated with hypochlorhydria, is also causal in acne. Intestinal dysbiosis and an overgrowth of bacteria in the small intestine, known as small intestinal bacterial overgrowth (SIBO) can lead to the production of toxic metabolites, increased intestinal permeability, and systemic inflammation [86].

Psoriasis

Psoriasis is a chronic inflammatory skin disease that is estimated to affect 2-3% of the population [87]. It is characterized by raised, erythematous plaques with silvery scales that can be associated with or without pruritus commonly present over extensors. The etiology of psoriasis is largely multifactorial involving genetics, environmental triggers and immune dysregulation. This leads to activation of pathways that result in elevations in pro-inflammatory cytokines. At the genus level, the results from systematic review states that Corynebacterium, Staphylococcus, and Streptococcus are reportedly more present in lesional skin, whereas a decrease in Cutibacterium is observed [88]. A lower abundance of S. epidermidis and C. acnes may also enable an increased colonization of S. aureus. Indeed, S. aureus colonization has been found to stimulate Th17 polarization in mice, suggesting that S. aureus triggers IL-17-mediated skin inflammation [89]. A clinical subform of psoriasis, called guttate psoriasis, is usually triggered by a streptococcal throat infection and generally evolves into the vulgaris (plaque) form which suggests a possible role of antibiotics in the disease process. The gut microbiome is believed to be involved in the development of pro-inflammatory Th17 cells, allowing it to modulate inflammation in diseases such as inflammatory bowel disease and obesity. Two studies reported lower relative abundance of Bacteroidetes and higher Firmicutes in psoriasis patients compared to healthy controls [90]. while Tan et al [91]. found a significant decrease in Akkermansia muciniphila which is believed to strengthen the integrity of the gut epithelium and protect against systemic inflammatory diseases like inflammatory bowel disease, obesity, and atherosclerosis. The barrier Integrity in psoriasis adheres not only to the skin but also at the intestinal level. Structural aberration in the form of decreased surface in the jejunum, lactose intolerance etc was reported in psoriasis patients and associated with the severity [92]. Recently, an oral derivative of a single strain of Prevotella histicola was tested for psoriasis and was found to be effective in murine models, confirmed in a phase 1b trial in humans but the results remain to be published [93].

Seborrhoeic Dermatitis

Seborrheic Dermatitis is a common inflammatory dermatosis that occurs in sebaceous gland bearing areas such as scalp, face and trunk. The incidence of the disease changes according to hormonal changes in the sebum. In addition to being associated with sebum-rich hair-bearing areas, seborrheic dermatitis is associated with Malassezia, which is a ubiquitous fungus and is a normal part of the human skin microbiome. It exists as 3 species namely Malassezia restricta, Malassezia furfur, and Malassezia globosa. Malassezia is lipophillic and usually colonises scalp and are the most abundant yeast species of the skin mycobiome [94]. The organism causes an overproduction of oleic acid,that disturbs the stratum corneum cells and evokes an inflammatory response on the scalp ,resulting in irritating free fatty acids and other metabolites producing sebaceous secretions on

the scalp, which in turn leads to an inflammatory response that results in skin changes [95]. A clinical study on probiotic strain Lactobacillus paracasei found significant improvements in severity and symptoms of moderate to severe dandruff compared to a placebo treatment [96].

Alopecia Areata

Alopecia areata is an autoimmune condition characterized by destruction of hair follicles. Activation and induction of the Th1 Lymphocytes leads to production of pro-inflammatory cytokines like IFN- γ that disrupts the anagen growth phase, ultimately resulting in hair loss and other manifestations of AA. Analysis of the scalp microbiome of patients with alopecia areata demonstrated an increase in C. acnes in combination with a decrease in S. epidermidis. A disbalance in Cutibacterium /Staphylococcus spp potentially play a role in alopecia areata [97]. Genes that are related to alopecia areata may also affect gut colonization with microorganisms that induce a Th1 response, which leads to the production of IFN-*g*, as IFN-*g* signals through a JAK/signal transducer and activator of transcription (STAT) signal pathway [98]. Induction of this pathway can cause abnormal growth of hair follicle cells and can even progress into hair loss.

Rosacea

Rosacea is a common inflammatory dermatosis characterised by erythema, papules and pustules. The skin of rosacea patients regularly contains an overgrowth of commensal skin microorganisms. Higher concentrations of Demodex folliculorum were detected, which usually inhabit the sebaceous glands. Cell-membrane components of the Demodex mite activates TLR2 which triggers KLK5 activity. The relative abundance of C. acnes decreased with advancing age however the severity of rosacea might increase with the relative abundance of Snodgrassella alvi, Geobacillus and Gordonia [99]. A link between gut microbial dysbiosis and rosacea has been hypothesised, as there is an increased risk of gastrointestinal disorders in rosacea patients [100] especially Helicobacterpylori infection (HPI) has been associated with the disease [101]. A higher abundance of Acidaminococcus and Megasphaera and a lower abundance of Peptococcaceae and Methanobrevibacter were reported [102].

Conclusion

The new era today is a research era which requires a close understanding of the subjects for the betterment of the patients. One of such, is the unique pathophysiological and relating mechanisms that abide the gut and skin microbiome. The multifactorial etiology pertaining to the dysbiosis can be any compound that has the ability to alter the gut epithelium or they can be merely the catabolic end products of metabolism. Hence understanding this skin-gut relationship is rewarding diagnostically as well as therapeutically to the physicians and dermatologists. The process in turn drives several other neuroendocrinological and pathologic mechanisms that are essential for a general wellbeing. A thorough review of this symbiosis can provide a future outlook to the emerging modalities that would specifically target several aspects of etiopathogenesis and serve as alternative to conventional and recalcitrant treatments that are undertaken for various dermatological disorders. Skin health and issues are ignored sometimes due to lack of a robust evidence based supportive treatments available. The evolving research topic has settled into many minds as to how we should and can, make use of these tiny creatures for diagnosing as well as treating the dreaded diseases. Our synopsis was an attempt to fill the lacunae in the research as well to compile what is already known.

Abbreviations

DCs: Dendritic cells; IL: Interleukins; AMP-Antimicrobial peptides; MHC-Major Histocompatibility complex; SCFA –Short chain fatty acids

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